# THE STUDY OF TROPONIN I IN SUPRAVENTRICULAR TACHYARRHYTHMIAS IN NON CAD PATIENTS

*Dissertation submitted in partial fulfillment of*

*The requirements for the degree of*

## M.D. (GENERAL MEDICINE)

**BRANCH - I**

**DEPARTMENT OF GENERAL MEDICINE**
**KILPAUK MEDICAL COLLEGE**

**CHENNAI – 600 010.**


**THE TAMIL NADU**

**DR.M.G.R. MEDICAL UNIVERSITY,**

**CHENNAI**

## APRIL - 2013

# BONAFIDE CERTIFICATE

This is to certify that the Thesis- "**THE STUDY OF TROPONIN I IN SUPRAVENTRICULAR TACHYARRHYTHMIAS IN NON CAD PATIENTS"** is a genuine work done by **Dr.R.HEMA,** Post-graduate student in Department of Medicine, Government Medical College, Kilpauk, under our guidance

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Finally, I would like to owe a lot to my patients for their support and without them this project would not be possible.

# DECLARATION

I, **Dr.R HEMA**, solemnly declare that the dissertation titled ““**THE STUDY OF TROPONIN I IN SUPRAVENTRICULAR TACHYARRHYTHMIAS IN NON CAD PATIENTS”** has been prepared by me.

This is submitted to the Tamil Nadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MD degree Branch I (General Medicine).

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Date:

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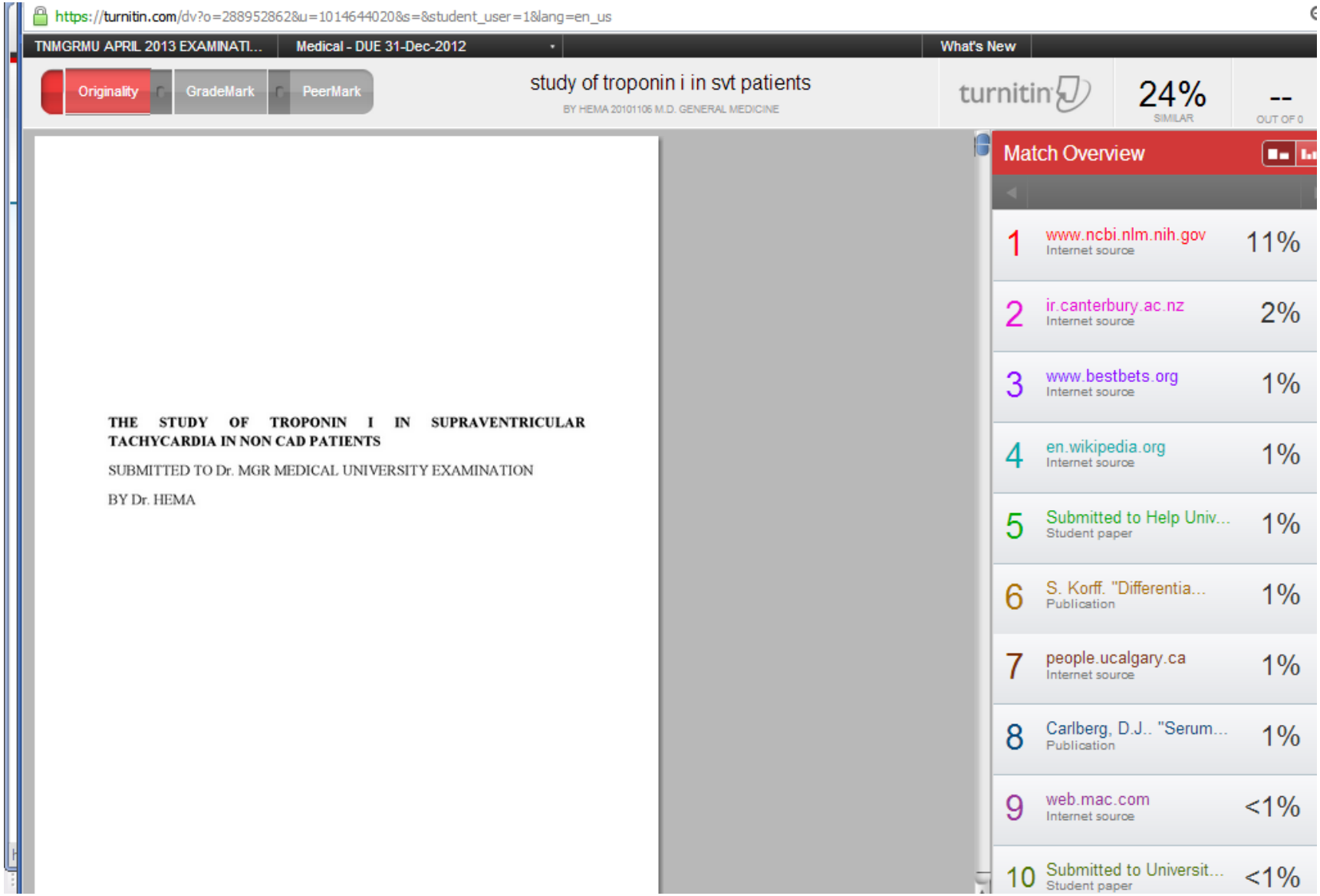
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# INTRODUCTION

According to American college of cardiology, the definition of myocardial infarction is elevation of cardiac enzymes (CK-MB, TROPONIN I, TROPONIN T) in the presence of symptoms(chest pain, palpitation, sweating or angina equivalents) with evidence of MI by ECG or ECHO.[1]Compared to Creatinine kinase , Troponins are highly specific for myocardial injury. By using third generation immunoassays, troponins have high sensitivity in detection of ischaemia. Hence, besides MI there are some acute conditions with elevated troponins like tachyarrhythmias, acute cerebrovascular accident, acute pulmonary thromboembolism, chronic kidney disease, septic shock, myocardial injury . But whatever the pathogenetic mechanism which leads to release of enzymes from myocyte, the elevated enzyme levels always indicates a poor prognosis in these cases[4]

Paroxysmal supraventricular tachycardia[5] (PSVT), a commonest form arrhythmia seen in the emergency department. Compared to ventricular tachyarrhythmias these patients are hemodynamically stable and they have good prognosis on treatment. Serum cardiac troponin I[6]

(cTnI) testing provides important risk stratification in these group of arrhythmias.[5]

In a retrospective study conducted by Calberg et al, he studied 51 PSVT patients[7] who were identified on the basis of INTERNATIONAL CLASSIFICATION OF DISEASES , NINTH EDITION codes. Fifty one were included in the data analysis. They had atleast one serum cTn value measured. Among them 11 had positive values (0.04 ng/ml)[8]. Six weeks outcome for these patients were studied. The results were one had recurrence, no deaths and moreover cTn I testing didn't identify PSVT patients at risk of worse prognosis[9].

Hence the recommendation of this investigation in PSVT patients needs to be studied in a large basis to ascertain it's importance in assessing the risk in these group of patients. [10]

Hence to test the hypothesis and to identify the association between the cardiac troponins and the non coronary SVT and its impact on the prognosis of the patient ,this study was conducted in our hospital

# REVIEW OF LITERATURE

## CARDIAC TROPONINS

The troponins are the proteins present in the myocyte. The troponin present in cardiac muscle is different from the troponin present in skeletal muscle.

The troponin in the cardiac myocyte consists of three units C, I, T[11] and is located on the thin (actin) filament of striated (cardiac) muscle. Troponin is a component of actin filaments alon g with actin and tropomyosin[12]. When calcium attaches to Troponin C, tropomyosin moves and exposes the active site of actin filaments, so that myosin head can attach to the actin and produce contraction of myocyte with utilization of ATP. When the calcium is absent, tropomyosin attaches with the active site of Actin, so that myosin can't contact with Actin.

Hence contraction is prevented. It has been shown in some studies that troponin I found to inhibit angiogenesis

The functions of various subunits are

Troponin C binds to calcium ions to produce a conformational change in TnI so that Tropomyosin inhibition is removed[12]

Troponin T binds to tropomyosin, to form a troponin-Tropomyosin complex[13]

Troponin I binds to actin in myofilaments to hold the troponin-tropomyosin complex in place[14]

Smooth muscle does not have troponin.

The cardiac isoforms of troponin T and I are only expressed in cardiac muscle. Hence, they are more specific than creatine kinase (CK) for myocardial injury[15]

Cardiac troponin I - it's molecular weight is 23KDa[16] and it's electron microscopic structure is 3 dimensional with ribbon like pattern. It's made up of 209 amino acid residues. The theoretical pI of cTnI is 9.87[17]. The amino acid in 22 and 23 position are serines. In vivo these amino acids can be phosphorylated by protein kinases, so we can see four forms of protein – onedephospho, two monophospho and one bisphospho within the cell. These biochemical Phosphorylation of

troponin changes the structural conformation of these protein and modifies its interaction with other troponins as well as it's binding with anti-TnI antibodies.[18]

Cardiac troponin T is 35 kda protein

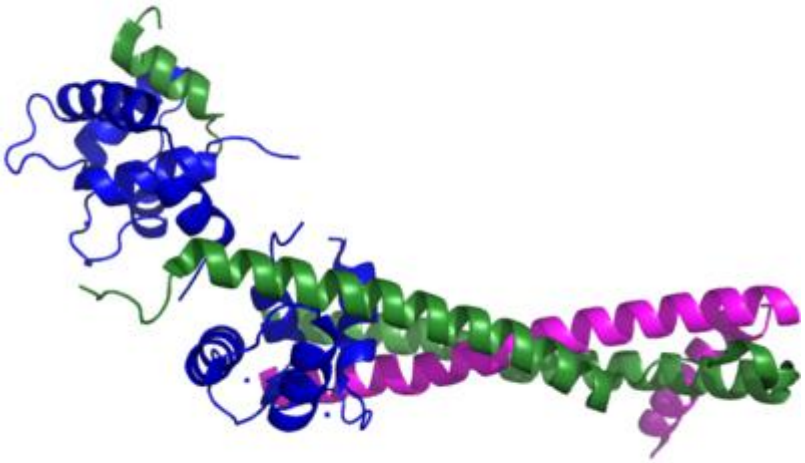


**FIG 1: ELECTRON MICROSCOPY STRUCTURE OF TROPONIN I**

Cardiac troponins were available as both bounded proteins within thick and thin filaments and as a free form within in the cytoplasm. These free forms constitutes 6–8% for cTnT and 3.5% for cTnI[19]. Following myocardial necrosis, cTn egress rapidly from the cell and will appear in blood after 2–4 hour and persists for up to 10–21 days

for effective diagnosis[20]. Further the detection of even minimal amount cardiac troponins by immunoassays is easy now, and it becomes cost effective, and the results are available readily, making them ideal biomarkers of myocardial injury

## VARIOUS CAUSES OF ELEVATED TROPONINS AND IT'S PREVALENCE

Various studies conducted previously in various countries have found causes for elevated troponin apart from ACS. In ACS its elevation should hav 100% prevalence to define myocardial infarction.

## CARDIAC TROPONIN RELEASE UNRELATED TO ACS

### SEPTIC SHOCK / SIRS

Sepsis or systemic inflammatory response syndrome (SIRS) contributes the major mortality group in intensive care units.The elevated cTnI (0.1 ng/ml) have been detected in 30% to 80%[21] of these cases in the acute phases. The cause for this significant elevation mainly due to the different etiology and multiple pathogenetic mechanism activated in sepsis. Among these cases, significant coronary artery disease has been ruled out by doing various investigations.

The reason for the release of cTn from myocardial cells might be a damage to the cell membrane caused by ischaemia, necrosis due to an oxygen supply–demand mismatch prevailing in the myocardium[22]. Due to fever and tachycardia the oxygen demand of the myocardium is increased whereas oxygen supply of the myocardium is reduced due to systemic hypoxemia from respiratory insufficiency, microcirculatory dysfunction due to lactic acidosis, hypotension . In addition, there will be activation of local and systemic inflammatory pathways leads to pouring of cytokines esp. tumour necrosis factor α, interleukin 6 , as well as bacterial endotoxins (Lipopolysaccharides), which leads to direct cytotoxic effects on myocardium and vascular endothelium[23].

Moreover, elevation of these proteins above normal limit provide prognostic significance and the extent of cTn elevation seems to correlate with the severity of the disease process. Ver Elst et al clearly demonstrated that cTnI - positivity in the sepsis patients was strongly associated with LV - dysfunction[24]. 78% of cTnI positive patients had LV dysfunction compared to 9% in cTnI - negative patients[25]. cTnI values were found to correlate significantly with the degree of hypotension and APACHE II score in crically ill patients.

Spies and colleagues et al studied 26 patients with sepsis, the group of patients with cTnT values ⩾ 0.2 μ/l showed an increased mortality rate (83% ) compared to the group with cTnT values below this value( 38%).[26]

**Pulmonary embolism**

Among 38 patients with Pulmonary thromboembolism, elevated cTnI values (> 0.4 ng/ml)[27] were seen in 47% of cases and elevated cTnT(0.1 ng/ml) were seen in 38% of cases. Every one in two patients presenting with chest pain and breathlessness diagnosed as PE had elevated cardiac enzymes in their biochemical profile. The reason for myocardial damage is excessive Right ventricular wall stress and strain due to sudden afterload increase to right ventricle due to elevated pulmonary pressure[28]. Other reasons may be reduced right coronary perfusion, hypoxia from perfusion–ventilation mismatch in thrombosed areas, systemic hypotension.

Studies which where done previously showing elevated troponins in PE mainly concludes 2 facts. First the peak cardiac troponin values in these patients are in lower cut off values and lasts only for shorter duration in blood. The second the brief appearance of this minimal amount of these troponins indicates only reversible myocardial injury

compared to high and long lasting troponins in acute coronary syndrome[29].

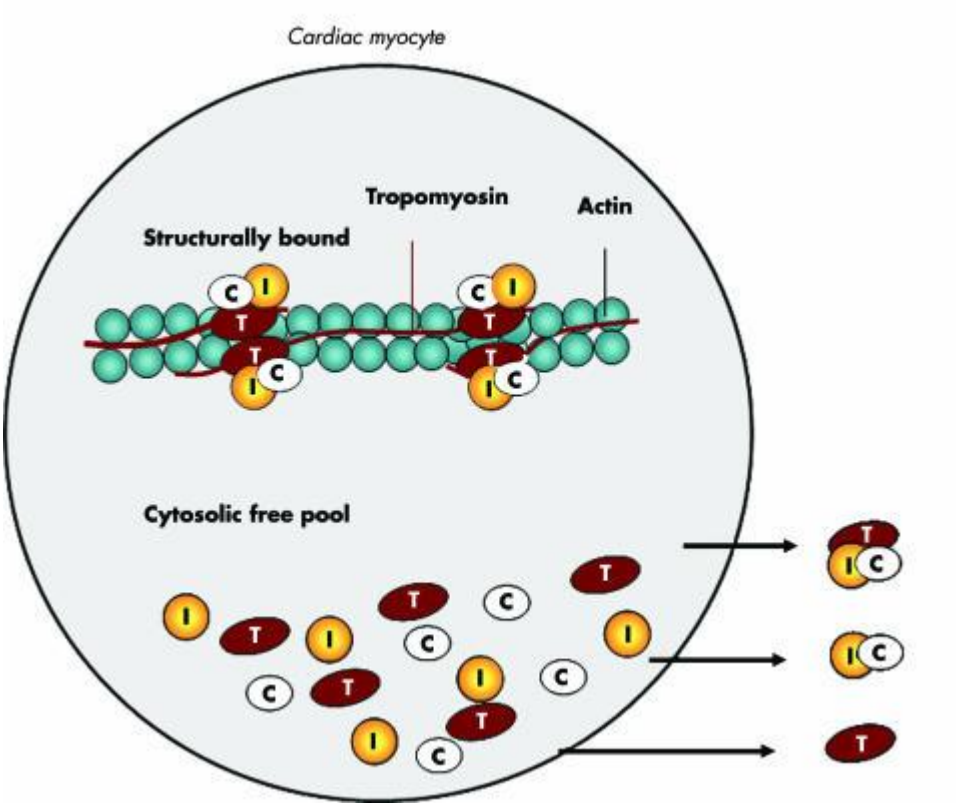



**FIG 2 : RELEASE OF CARDIAC ENZYMES FROM CYTOSOLIC POOL**

**Acute and chronic heart failure**

Elevated cTn in heart failure (HF) indicates decreased left ventricular ejection fraction and correlate well with the severity of heart failure and prognosis[30]. The major pathogenesis were progressive myocyte loss due to wall tension created by high filling pressures. The co

existing features are renin –angiotensin- aldosterone axis activation, overt sympathetic activity ,activation of inflammatory pathway which contributes further to myocardial damage[31]. Sometimes wall tension will aggravate sub endocardial ischaemia leading on to severe chest pain which mimicks ACS.

In patients with chronic stable HF, elevated cTnI values were found in 15–23% of cases (> 0.1 ng/ml). For cTnT, values above 0.1 ng/ml were reported in 10–15% of cases[32]. There was no difference between the ischaemic and non-ischaemic group. In acute HF, 52–55% had elevated cTnT values. The presence of cTn in HF predicts a worse prognosis compared to troponin negative ones[33]. Patients with increased troponin values have usually shown ECHO with lower ejection fractions, grade iii and grade iv HF (New York Heart Association functional class), and poorer outcome. Serial measurements of cTn was mandatory in grade iii and grade iv heart failure as it provides additional prognostic information. A decrease of cTn from the previous values usually was associated with improvement of symptoms, left ventricular function and ejection fraction while persistently elevated or rising troponin values indicates high mortality.

**Strenuous exercise**

Following prolonged isometric exercise, there will be elevation of cardiac troponins which will be normalized within 24 hours.This was different from the elevation seen in ACS. several studies were done previously to rule out association between the elevated enzymes and it's prognostic significance. None of them establishes their relationship. Further studies are needed to clarify some inconsistencies in this area.

**Acute pericarditis/myocarditis**

Acute pericarditis is commonly diagnosed in patients with acute chest pain with ECG showing persistent concave ST elevation. The most confusing issue was the presence of elevated enzyme level which makes us to diagnose this as ACS. Although troponins are not present in the pericardium, cTnI was reported to be elevated in 32–50% of cases of pericarditis[34], as a consequence of the involvement of the epicardium in the inflammatory process.

In patients with acute myocarditis , the only significant finding in ECG was inappropriate sinus tachycardia with ST-T changes .cTnI concentrations have been found to be increased in 34% of patients. The coronary angiogram was done in certain group to rule out ACS. The gold

standard for diagnosis was endomyocardial biopsy which shows myocytolysis and lymphocytic infiltrates in myocytes. Even this was positive only in 10% 0f cases.[35] Hence elevated troponin values are more sensitive in diagnosis compared to other diagnostic modality

**Cardiotoxic chemotherapy**

The cardiotoxic side effects of chemotherapy were well known to all. The commonly known adverse effects following chemotherapy noted are ischaemia, endomyocardial fibrosis, cardiomyopathy, pericarditis and different types of arrhythmias due to long QT syndrome36. So these patients are routinely investigated based on risk factors. Recent studies showed that biochemical markers like natriuretic peptides, creatinine kinase, troponins are found elevated in subset of patients who are at risk of myocardial damage[37] So their role in predicting myocardial damage needs evaluation in future.

**External Current Cardioversion/Defibrillator Shocks**

Radiofrequency ablation used in certain arrhythmias to cut the closed electrical circuits, causes an elevation of cTn in more than 90% of patients and is due to direct myocardial injury[38], but these usually have no prognostic utility. External current cardioversion (ECV) used in

cardiac arrest and asystole caused small increase of cTnI and no increases of cTnT, especially when biphasic mode was used compared to monophasic mode[39]. Repetitive defibrillator shocks given in advanced life support procedures, polymorphic VT, VF are known to cause release cTn into the blood. But diagnostic and prognostic significance was not made out till now.

**Infiltrative Disorders**

In systemic amyloidosis, the presence of cardiomyopathy indicates worst prognosis. Dispenzieri and colleagues studied 261 patients with amyloidosis. He showed that the median survival of patients with elevated troponins was significantly reduced[40] (six months compared to that of patients with normal values (22 months).

The main pathogenesis was extracellular amyloid deposition causes compression of myocytes with subsequent release of cytosolic pool of cTn[41]. In many patients, the effective diagnosis of cardiac involvement is made incidentally by a positive troponin values in the absence of signs or symptoms of ACS. So it is mandatory to do troponin assays in all amylodosis patients with significant systemic involvement which will allow earlier detection of prognostically adverse cardiomyopathy.[42]

**Post-heart transplantation**

Following transplantation, the presence of elevated cTn indicates to the possibility of allograft rejection apart from cardiac failure. In 1998 Dengler and colleagues found that cTnT values increased in direct proportion with the severity of graft rejection according to the ISHLT (International Society of Heart and Lung Transplantation) grading system[43]. In the patients with severe rejection (ISHLT grade 3 and 4), almost all patients showed elevated cTnT values. Hence if cTnT was negative, significant rejection could be excluded with a specificity of 92%[44]. Recently the investigation of choice for allograft rejection is endomyocardial biopsy. If cardiac troponins are negative, Rejection can be ruled out easily.[45]

**Myocardial contusion**

In severe blunt injury chest, cardiac contusion contributes 3–56% of cases. Cardiac troponins were found to be significantly elevated in 15–45% of patients[46] with cardiac contusion. The absence of these markers in blood along with echocardiography, we can easily exclude major contusion in the heart except in few cases.

## RENAL FAILURE/END STAGE RENAL DISEASE

### Troponins in renal failure with ACS

In CKD patients with ACS, troponin values are elevated similar or just higher compared to ACS in non CKD patients. However, in patients with chronic kidney disease, cTn concentrations develop higher peak values and troponin remains higher[47] for longer duration (1 month). so repeated early measurements are needed to detect a acute rise in the already raised baseline values which indicates an acute ischaemia or infarction.

### Troponins in asymptomatic renal failure patients

Cardiovascular disease was the commonest cause of death in patients with ESRD, hence a clinically silent cardiac pathology can always underlie these troponin elevations48. Previous studies were analysed based on the prevalence and prognostic impact of cTn in patients with ESRD either on haemodialysis or renal replacement therapy. Both cTnT and cTnI are commonly increased in asymptomatic patients with ESRD, even when there is no suspected myocardial ischaemia[49]. This means they have elevated baseline values compared to non CKD patients.

Using third generation assay for cTnT, up to 53% of haemodialysis patients had elevated cTnT values, while 19% of patients showed elevated cTnI values.[50] This finding led to significant therapeutic implication that cTnI would be a more sensitive marker for myocardial ischaemia in patients with CKD compared to cTnT[51]. The significant discovery is that troponin I undergoes fragmentation, phosphorylation, oxidation in CKD patients and was highly unstable so that they are undetectable by using routine immunoassays.

Several previous studies were analysed for this baseline elevation of cardiac troponins. The first possible explaination was re expression of these cardiac isoforms in skeletal muscle in CKD patients[52]. The other possible explainations are reduced renal clearance of troponin in CKD patients, possibly renal anemia which causes oxygen supply – demand mismatch. Moreover troponins undergoes metabolism in the blood to form smaller 8 – 25 kD proteins which were easily eliminated by normal kidney53. But CKD patients have decreased ability to remove this substances in blood leading to elevated baseline parameters in these patients.

Several large observational studies have clearly shown that elevated concentrations of cTnT in patients with ESRD are valuable, independent, short and long term predictors of cardiac death[54].

## ENZYMES IN SUPRAVENTRICULAR TACCHYARRYTHMIAS

### TABLE 1

| Author, date and country | Patient group | Study type (level of evidence) | Outcomes | Key results | Study Weaknesses |
|---|---|---|---|---|---|
| Redfearn, et al. 2005 UK | 3 patients ages 22-72 with SVT and elevated troponin levels. | Retrospective cohort. | Raised troponin I leading to coronary angiography: | All patients had normal coronary vessels on angiography. The 72 yo patient was also found to have evidence of hemodynamic compromise. | Limited patient group with no comparison group of patients with SVT and CAD. Database entry was retrospective and subject to bias. |
| Zellweger, M, et al. 2003 Switzerland | 4 adult patients ages 44-57 with SVT and elevated troponins. | Case series. | Raised troponin I leading to subsequent testing for CAD | All patients were determined to not have CAD as ruled out by echocardiography with gated myocardial perfusion (1 patient), stress echocardiography (2 patients), and coronary angiogram (2 patients). | Limited case series. |
| Bakshi, T.K., et al 2002 New Zealand | 3 patients ages 37-49 with SVT and elevated troponin levels. | Prospective cohort. | Consecutive patients with elevated troponins who underwent angiography for suspected coronary disease. | 2 patients had high likelihood of ACS according to electrocardiographic changes and 1 patient had low likelihood on ECG. All patients were found to have normal coronary arteries on angiography. | Limited case series. |

| Author, date and country | Patient group | Study type (level of evidence) | Outcomes | Key results | Study Weaknesses |
|---|---|---|---|---|---|
| Miranda, RC, et al 2006 Brazil | 49 year-old female with SVT | Case study. | Elevated troponin I level. | Patient was found to have normal coronary arteries on angiogram. | A case study. |
| Patanè, S, et al 2009 Italy | 49 year-old female with SVT | Case study | Elevated troponin level without clinical or ECG presentation of ACS. | On echocardiography mitral regurge and mild interventricular septal hypertrophy were present. ECG did not reveal any significant changes.Patient recovered over several days with a return to baseline troponin I levels. | A case study. |
| Yeo, KK, et al. 2006 United States | 3 patients ages 22-58 with SVT and elevated troponin levels. | Case series. | Elevated troponin I level. | All patients were found to have normal coronary arteries on coronary angiography. | Limited case series. |
| Kanjwal et al, 2008, USA | 7 patients ages 18-67 with tachycardias (5 with SVTs) and elevated troponin levels. | Case series | All patients presented with chest comfort and elevated troponin levels and subsequently underwent coronary angiography. | All patients underwent coronary angiography and were found to have normal epicardial vessels. There was no evidence of hemodynamic instability in any of the patients. | Limited case series |

## PATHOPHYSIOLOGY OF SUPRAVENTRICULAR TACCHYARRHYTHMIA

Supraventricular tachyarrhythmia is defined as narrow complex arrhythmia (QRS <120 msec) with heart rate of 180- 240 beats / min usually arises either from the atria or within AV node or accessory pathways connecting atria with ventricle. There are 2 types

1. AVNRT
2. AVRT

### Atrioventricular Nodal Reentrant tachycardia

Normally there are some differences in the electrical properties of the various tissue types in the AV node which are responsible for AV nodal reentrant tachycardia. There are 2 types of pathways in AV node one is a slow one and other is a fast one[55]. In typical AVNRT the conduction occurs via slow pathway to depolarize the ventricle .The retrograde conduction occurs via fast pathway to reactivates the atria to form closed circuit. In atypical AVNRT the conduction first occurs via the fast pathway and retrograde conduction via the slow pathway[56]

**Preexcitation Syndrome tachycardia or Atrioventricular reciprocating tachycardia**

In most patients who have reciprocating tachycardias they have accessory pathways. The accessory pathways conducts more rapidly than the normal AV node. After the ventricles have been excited, the impulse is able to enter the accessory pathway retrogradely and return to the atrium. A closed circuit loop of this type establishes the tachycardia. In wolf Parkinson syndrome there are 5 types of pathways[56]

**ORTHODROMIC**

In this group the antegrade conduction occurs via normal AV NODE retrograde conduction via accessory pathway giving rise to narrow complex long RP tachycardia

**ANTIDROMIC**

Sometimes the activation travels via the accessory pathway while the retrograde conduction is via the normal AV node producing wide QRS tachycardia

These accessory pathways detected by electrophysiologic studies

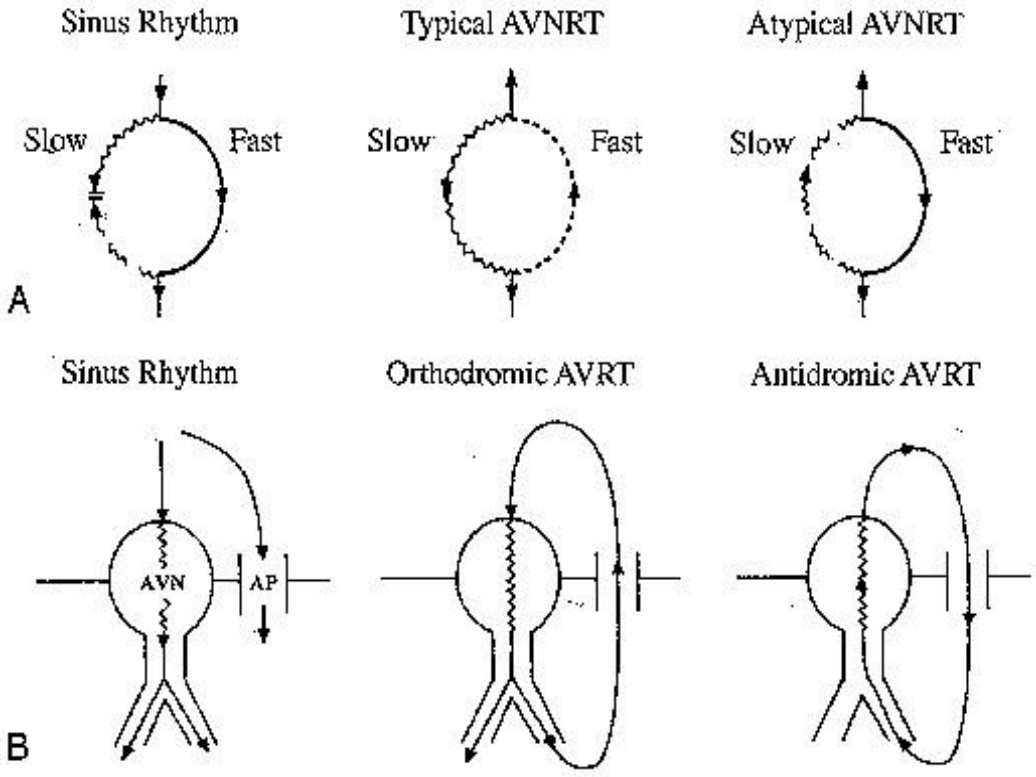



**FIG 3 : RE-ENTRANT PATHWAYS IN BOTH AVRT AND AVNRT**

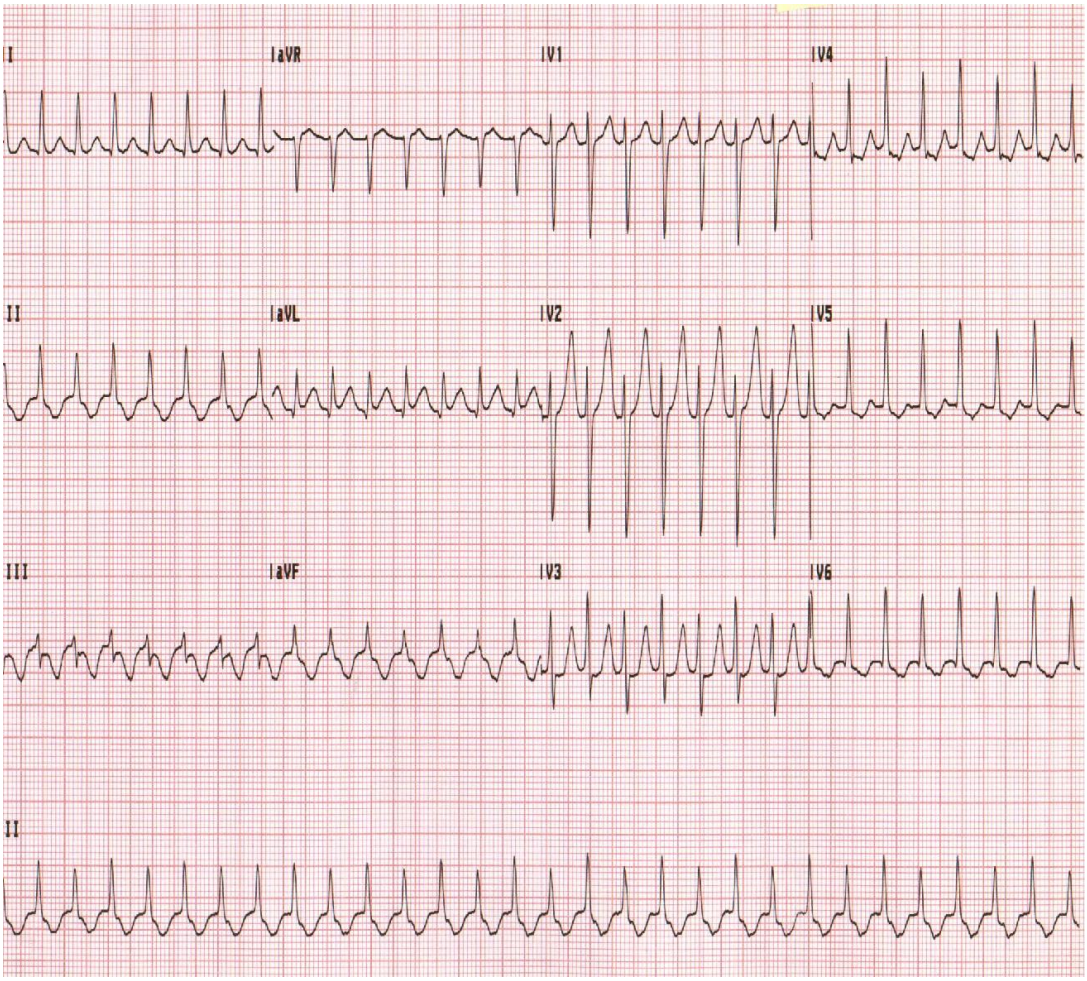



**FIG 4 : ECG SHOWING AVRT**

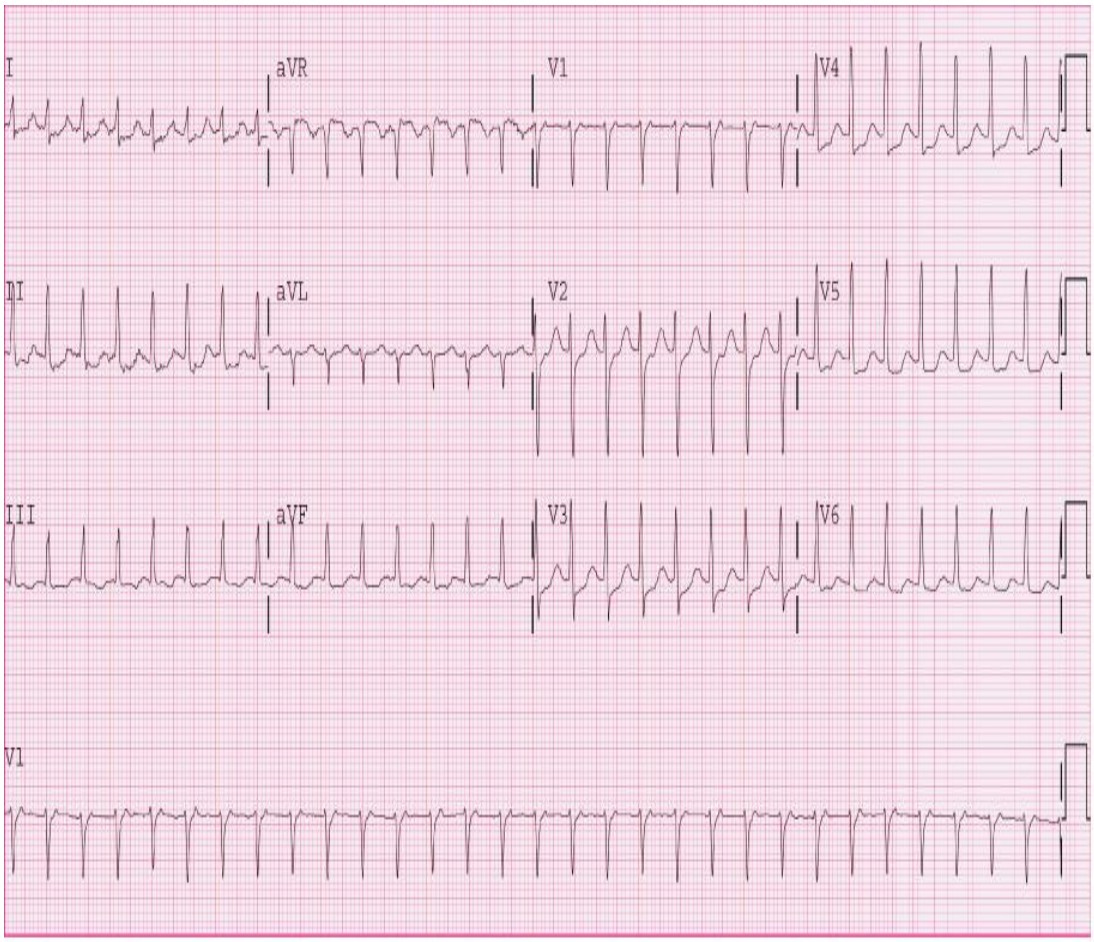
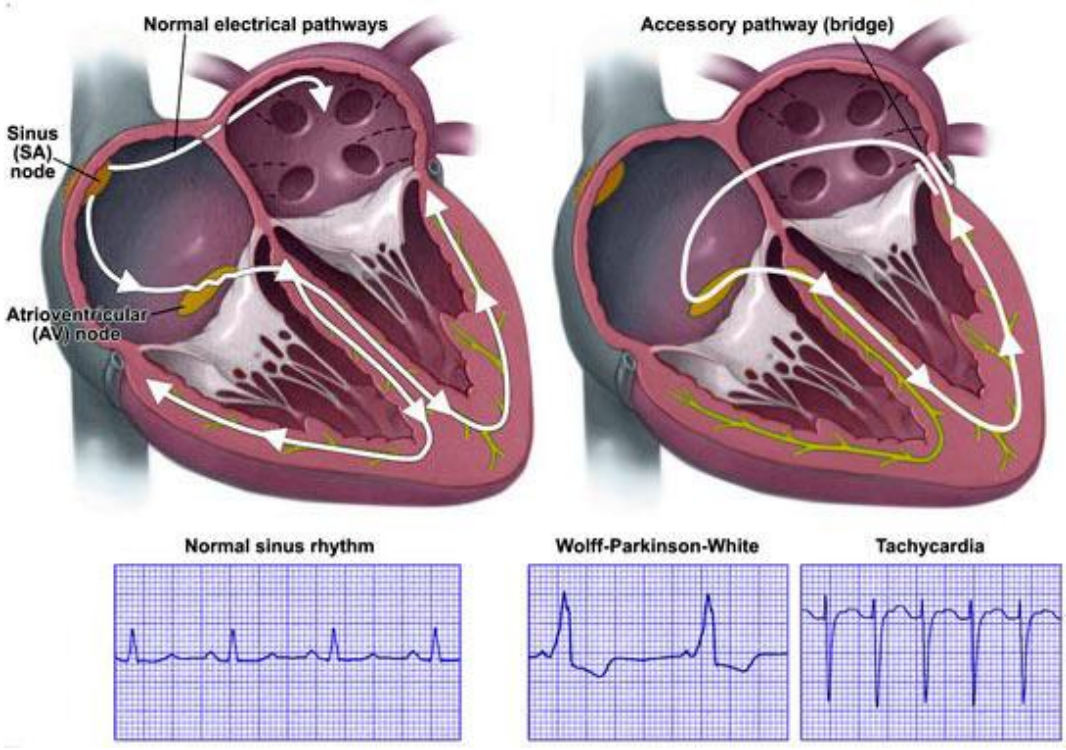
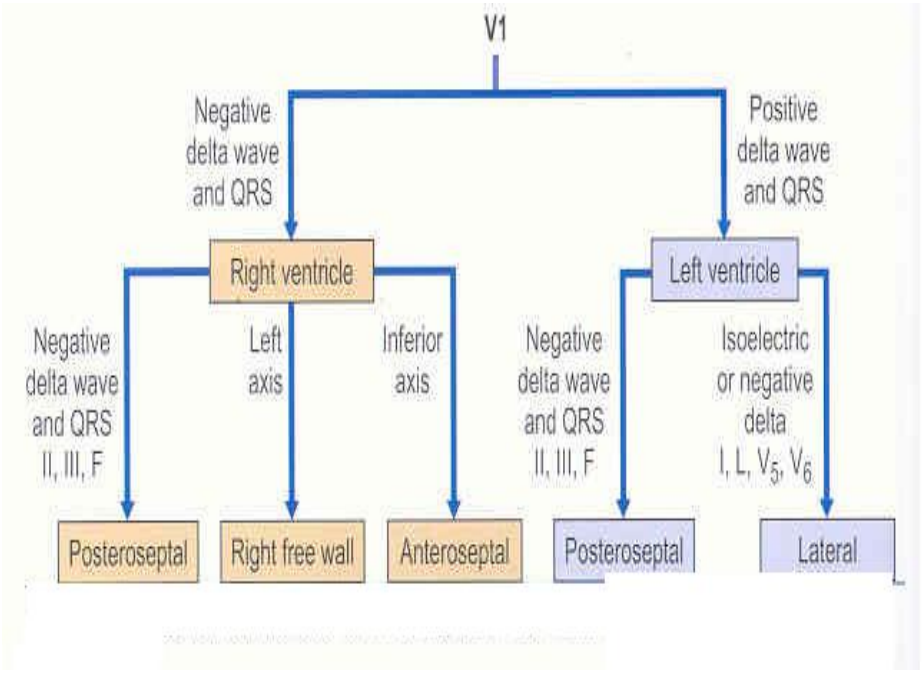



**FIG 5 : ECG SHOWING AVNRT**




**ECG FINDING IN 5 TYPES OF PATHWAY IN WPW SYNDROME**

## SIGNIFICANCE OF CARDIAC TROPONINS IN ARRHYTHMIAS

The repeated incidence of tacchyarrhythmias in these patients leads to leads to variable amount of myocardial injury leading to release of myofibrillar enzymes. Whatever the proposed mechanism for the enzyme elevation, the presence of significant troponins in the blood causes worse prognosis which indicates significant amount of myocardial damage had occurred already.

Repeated episodes of tachyarrhythmias

abnormal and incoherent myocardial contraction

shear stress on the myocardium and endocardium

ischaemia of endocardium, with release of myocardial cytosolic pool

leading on to reduced myocardial contractility

impending ventricular dysfunction

## CAN THIS BE USED AS A PROGNOSTIC INDICATOR

As there is minimal myocardial injury in the svt compared to ACS the utility of using it as prognostic marker needs consideration and further studies are needed to evaluate this in detail

## AS AN INDICATOR FOR IMPENDING CARDIAC FAILURE IN ARRHYTHMIA PATIENTS

There is only minimal and ill sustained elevation of this cardiac enzymes in arrhythmia hence it's utility as a marker for impending failure has to be considered. Moreover chronic heart failure patients have elevated cardiac enzymes and they are predisposed to variety of arrhythmias. Hence further prospective studies are needed in future to assess impending failure in arrhythmic patients

## FALSE POSITVE TROPONIN IN THE HEALTHY VOLUNTEERS

One percent of healthy volunteers who showed minor elevation of cTnT were considered as false positive after eliminating the possibility of ACS by coronary angiography[57]. In general practice the significance of elevated troponins were interpreted based on clinical findings to avoid unnecessary procedure in these healthy patients.

**Causes of false-positively elevated troponins[58]**

* Analyser or analyte malfunction

* The presence of microparticles.

* The presence of fibrin clots

* Rhabdomyolysis

* Heterophilic antibodies as seen in Paul bunell ‘s test

* Rheumatoid factor

# AIM OF THE STUDY

To study the clinical profile of non CAD supraventricular tachyarrhythmias

To investigate the relationship of serum creatinine kinase(MB) and trponin I in this patients.

To identify whether any association exists between serum cardiac enzymes and the following parameters.

Age

Sex

Blood pressure

Alcohol

Diabetes mellitus

Smoking

Obesity

Hemoglobin

Type of SVT

Presence of LVH/diastolic dysfunction in ECHO

# BACKGROUND

## SELECTION OF SUBJECTS:

Patients who admitted cardiac CCU department in Kilpauk Medical College Hospital with palpitation diagnosed as SVT by ECG after excluding exclusion criteria.

## INCLUSION CRITERIA:

Supraventricular tachycardia diagnosed by ECG with heart rate 180 to 240/min with regular narrow complex tachycardia either as AVRT or AVNRT

## EXCLUSION CRITERIA

Acoholic Liver Diseases

Cerebro vascular accident

Patients with chronic heart failure

Valvular heart disease

Chronic kidney disease

Malignancy

CAD with failure

Infiltrative disorder

Acute pulmonary embolism

Chronic or current infections

Use of anti-cancer drugs in the past 30days

# MATERIALS AND METHODS

Setting : Kilpauk Medical College

Study design:Descriptive analytical study

Period of study: 2010 May -2012 December

Sample size: 75 subjects

This study is to analyse the prevalence of supraventricular tachyarrhythmia due to non CAD causes and to analyse the various risk factors in them and to find the level of CK(MB) and troponin I in these patients . To study about any correlation exists between the various risk factors and the cardiac enzymes level.

**Cases are investigated by following measures.**

History –duration of CAD, symptoms, family history of CAD, smoking, alcohol intake, past history regarding the number of episode of SVT were asked.

General examination

Weight

Systemic examination

Blood pressure by sphygmomanometer

12 lead Electrocardiogram in rhythm strip

Complete blood count

Renal function test

ECHO

- In transthoracic ECHO LV dysfunction, LV hypertrophy ejection fraction, valve sclerosis, calcification, the presence of diastolic dysfunction are noted.

**ENZYME ANALYSIS**

Blood samples were taken immediately after admission and sent for assays. The cardiac troponins were measured using third generation immunoassays. Cardiac troponins are detected in the serum by the use of monoclonal antibodies to epitopes of cTnI and cTnT.

## IMMUNE ASSAYS

The continuous improvement of assay performance has reduced rates of false positive results over the years by eliminating heparin interference, or cross-reactivity with skeletal muscle. Sometimes first and second generation troponin assays often shows positive results in patients with severe skeletal muscle injury because of an unspecific binding of skeletal muscle troponin T to the test tube59. Using a third generation cTnT assay, we found no cross-reactivity between cTnT and CK, neither in exercise-induced skeletal muscle trauma nor after rhabdomyolysis.60

**NORMAL TROPONIN LEVELS LESS THAN 0.04 ng/ml**

**NORMAL CK(MB) LEVELS LESS THAN 5.5 ng/ml**

# DATA ANALYSIS

**AMONG THE 75 PATIENTS I HAVE STUDIED 52 OF THEM HAVE**

**ELEVATED TROPONIN VALUES AND 17 OF THEM HAVE**

**ELEVATED CK(MB) VALUES DURING THEIR ADMISSION**

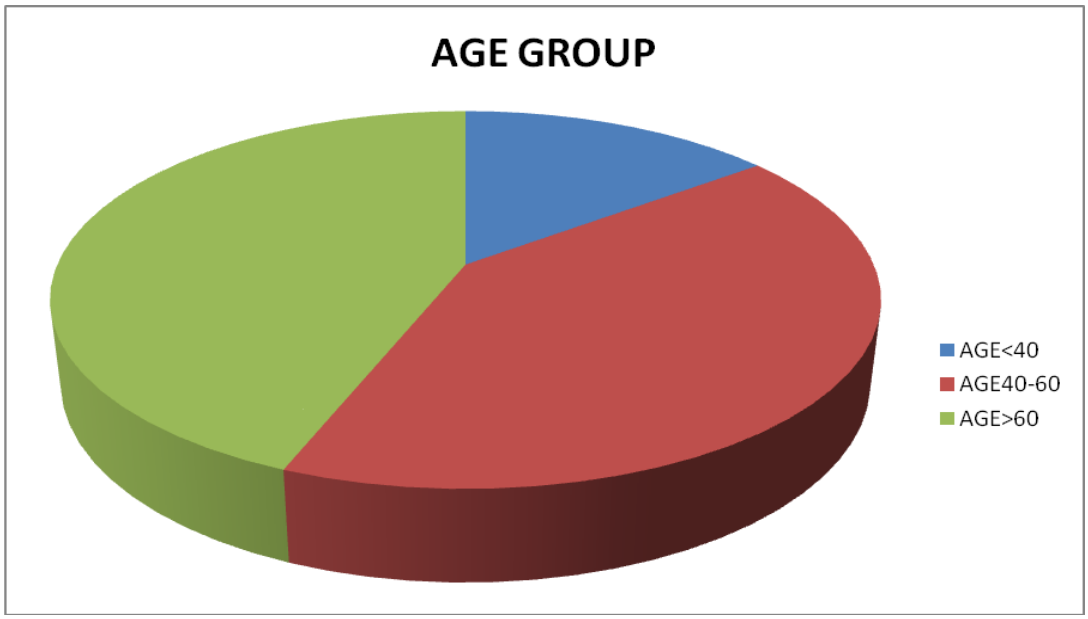



**FIG : 6**

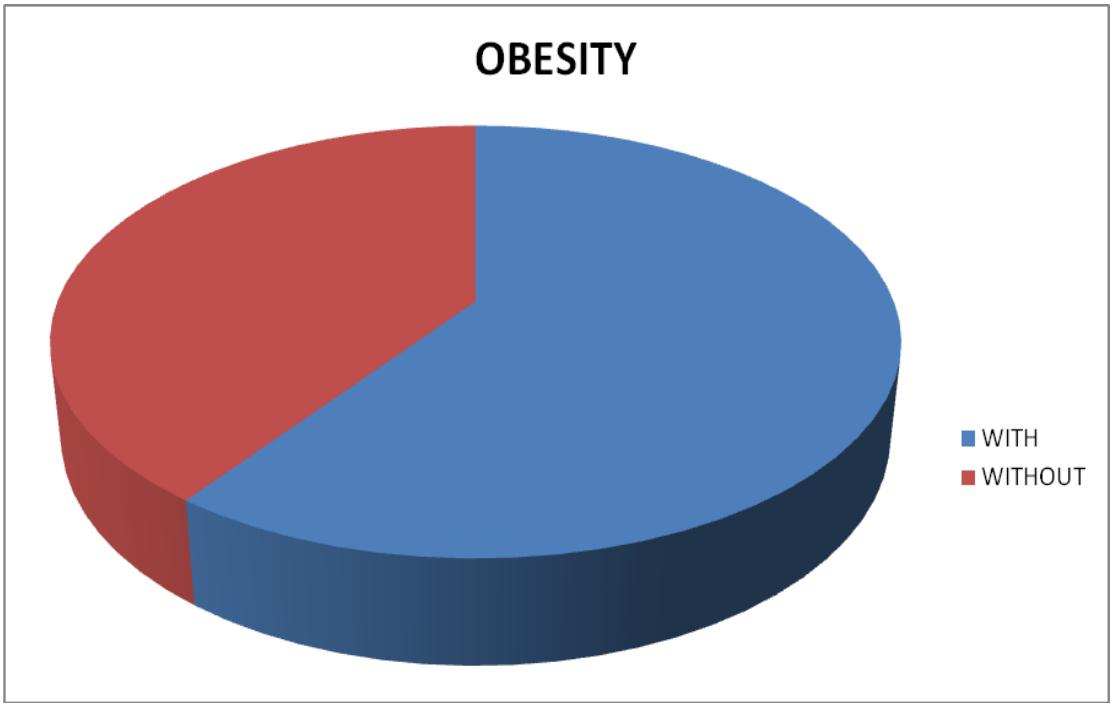



**FIG : 7**

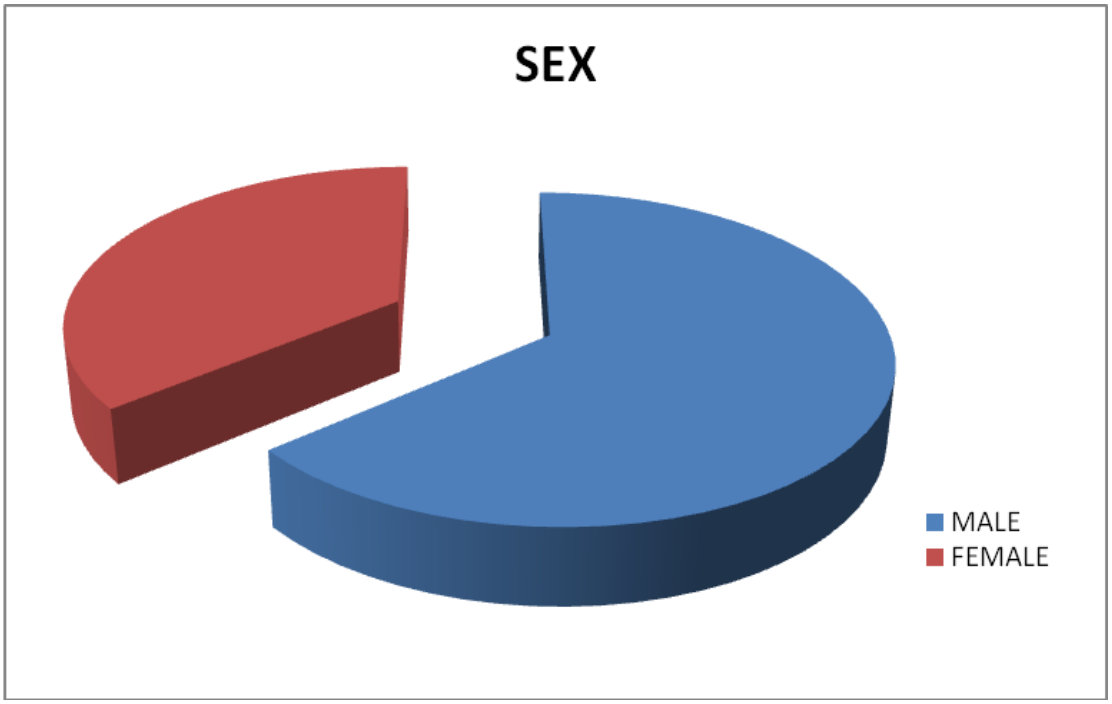



**FIG - 8**

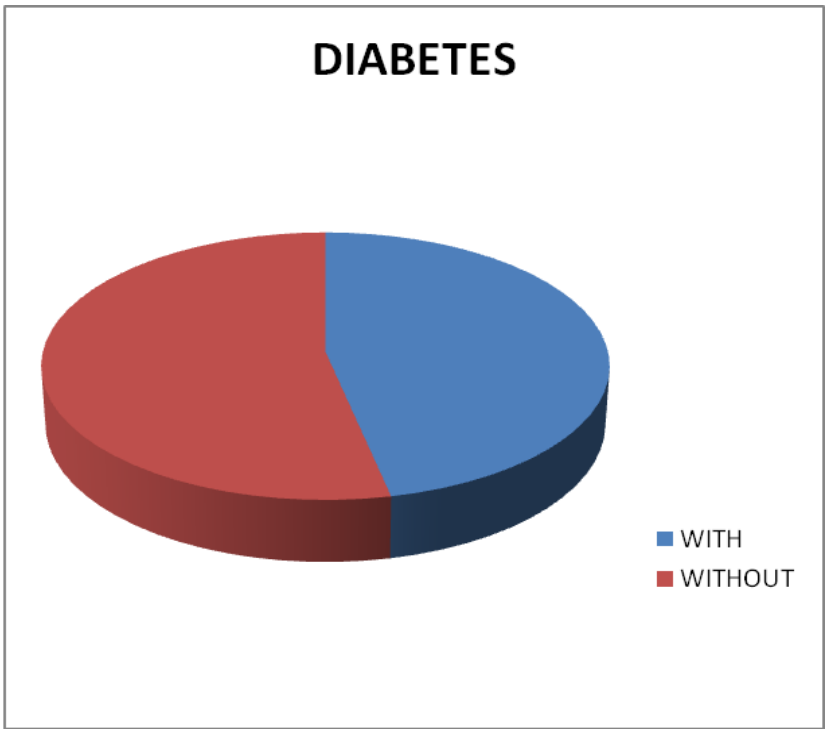



**FIG : 9**

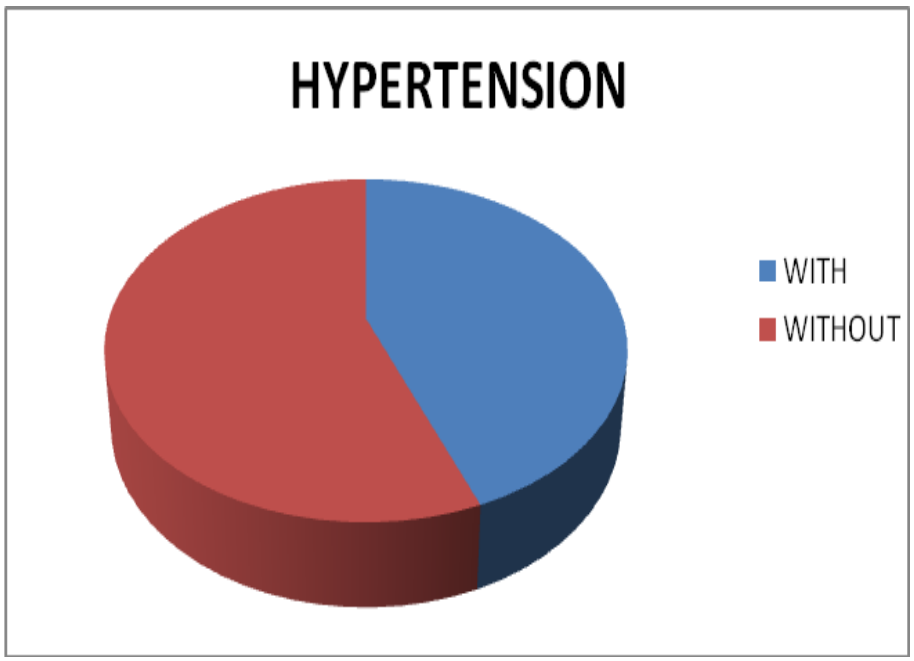



**FIG : 10**

**Descriptive Statistics**

Among the data collected the mean and standard deviation of various variables have been calculated

**TABLE - 2**

| | **Mean** | **Std. Deviation** | **N** |
|---|---|---|---|
| AGE | 57.07 | 15.302 | 75 |
| WEIGHT | 69.67 | 14.063 | 75 |
| HB | 10.636000 | 2.1198955 | 75 |
| CK-MB | 4.186400 | 2.5871453 | 75 |
| Tn-i | .607600 | .4996021 | 75 |
| sys BP | 128.48 | 25.923 | 75 |
| DIA BP | 81.76 | 11.745 | 75 |

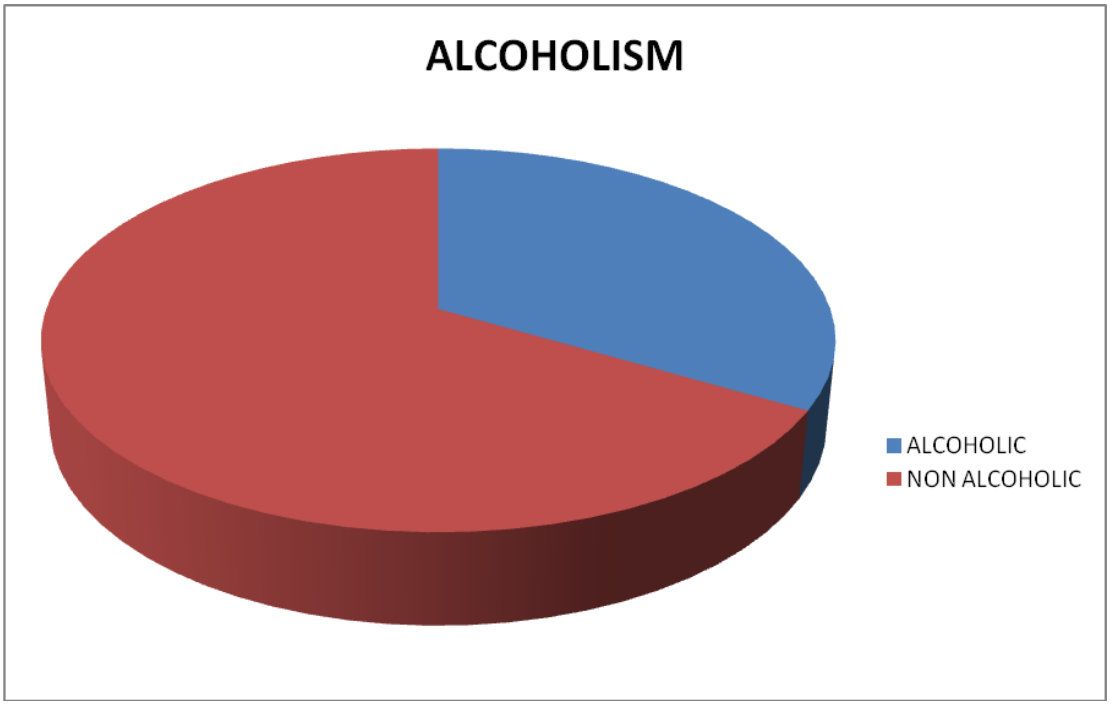

ALCOHOLISM
ALCOHOLIC
NON ALCOHOLIC

**FIG : 11**

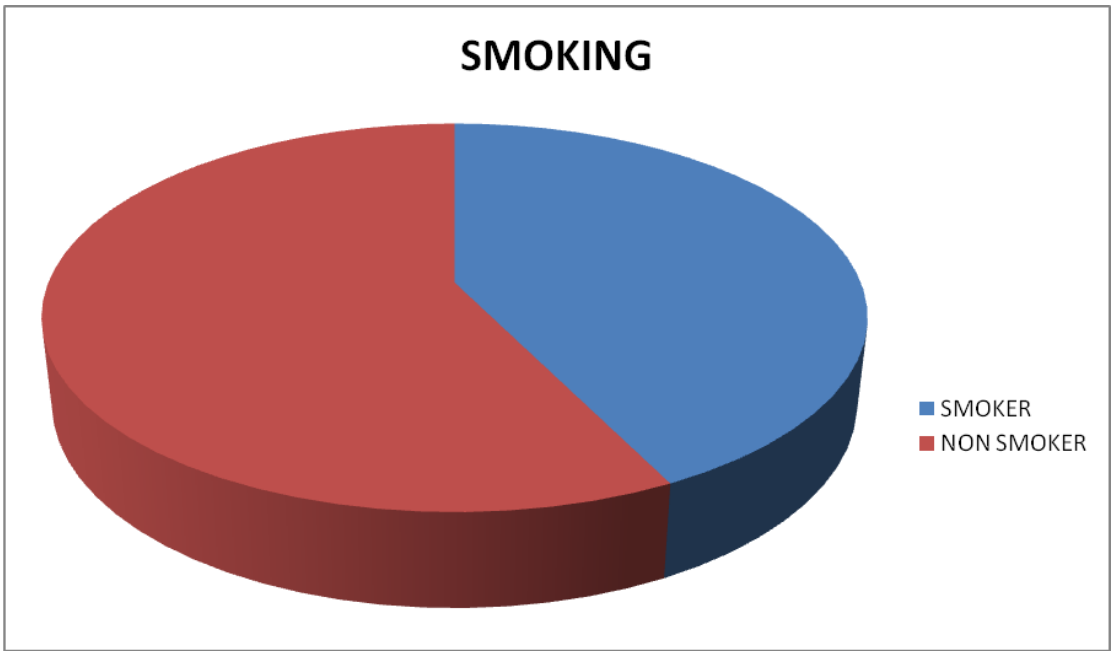

SMOKING
SMOKER
NON SMOKER

**FIG : 12**

| F | | AGE | WEIGHT | HB | CK-MB | Tn-i | sys BP | DIA BP |
|---|---|---|---|---|---|---|---|---|
| AGE | Pearson Correlation | 1 | .042 | -.276* | .283* | .142 | .195 | .120 |
| | Sig. (2-tailed) | | .717 | .017 | .014 | .226 | .093 | .307 |
| | N | 75 | 75 | 75 | 75 | 75 | 75 | 75 |
| WEIGHT | Pearson Correlation | .042 | 1 | .135 | -.111 | .035 | .254* | .100 |
| | Sig. (2-tailed) | .717 | | .248 | .341 | .767 | .028 | .395 |
| | 28N | 75 | 75 | 75 | 75 | 75 | 75 | 75 |
| HB | Pearson Correlation | -.276* | .135 | 1 | -.024 | .097 | .020 | .192 |
| | Sig. (2-tailed) | .017 | .248 | | .836 | .406 | .864 | .100 |
| | N | 75 | 75 | 75 | 75 | 75 | 75 | 75 |
| CK-MB | Pearson Correlation | .283* | -.111 | -.024 | 1 | .340** | .082 | .202 |
| | Sig. (2-tailed) | .014 | .341 | .836 | | .003 | .487 | .082 |
| | N | 75 | 75 | 75 | 75 | 75 | 75 | 75 |
| Tn-i | Pearson Correlation | .142 | .035 | .097 | .340** | 1 | .265* | .295* |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Sig. (2-tailed) | .226 | .767 | .406 | .003 | | .022 | .010 |
| | N | 75 | 75 | 75 | 75 | 75 | 75 | 75 |
| sys BP | Pearson Correlation | .195 | .254* | .020 | .082 | .265* | 1 | .612** |
| | Sig. (2-tailed) | .093 | .028 | .864 | .487 | .022 | | .000 |
| | N | 75 | 75 | 75 | 75 | 75 | 75 | 75 |
| DIA BP | Pearson Correlation | .120 | .100 | .192 | .202 | .295* | .612** | 1 |
| | Sig. (2-tailed) | .307 | .395 | .100 | .082 | .010 | .000 | |
| | N | 75 | 75 | 75 | 75 | 75 | 75 | 75 |

*. Correlation is significant at the 0.05 level . TABLE 3

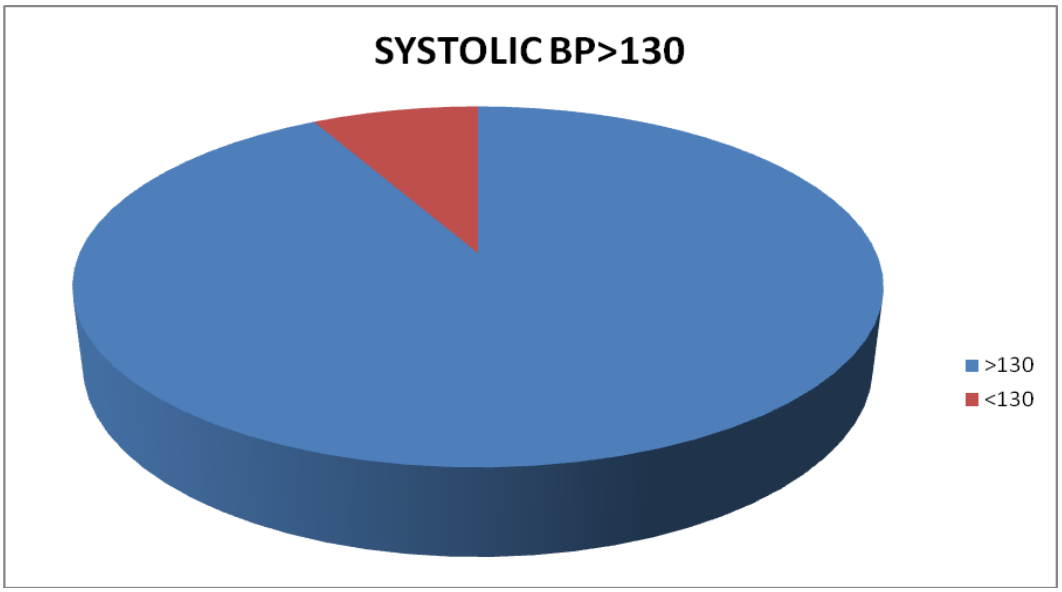



**FIG : 13**

**IN ABOVE TABLE THE CORRELATION BETWEEN VARIOUS VARIABLES HAVE BEEN SOUGHT OUT USING PEARSON CORRELATION TECHNIQUE**

| | |
|---|---|
| **CK-MB** | **AGE** |
| **WEIGHT** | **SYSTOLIC BP** |
| **HEMOGLOBIN** | **AGE** |
| **CK – MB** | **TROPONIN** |
| **SYSTOLIC BP** | **TROPONIN** |
| **DIASTOLIC BP** | **CK – MB** |
| **DIASTOLIC BP** | **TROP - I** |

Correlation was found between the above said parameters.

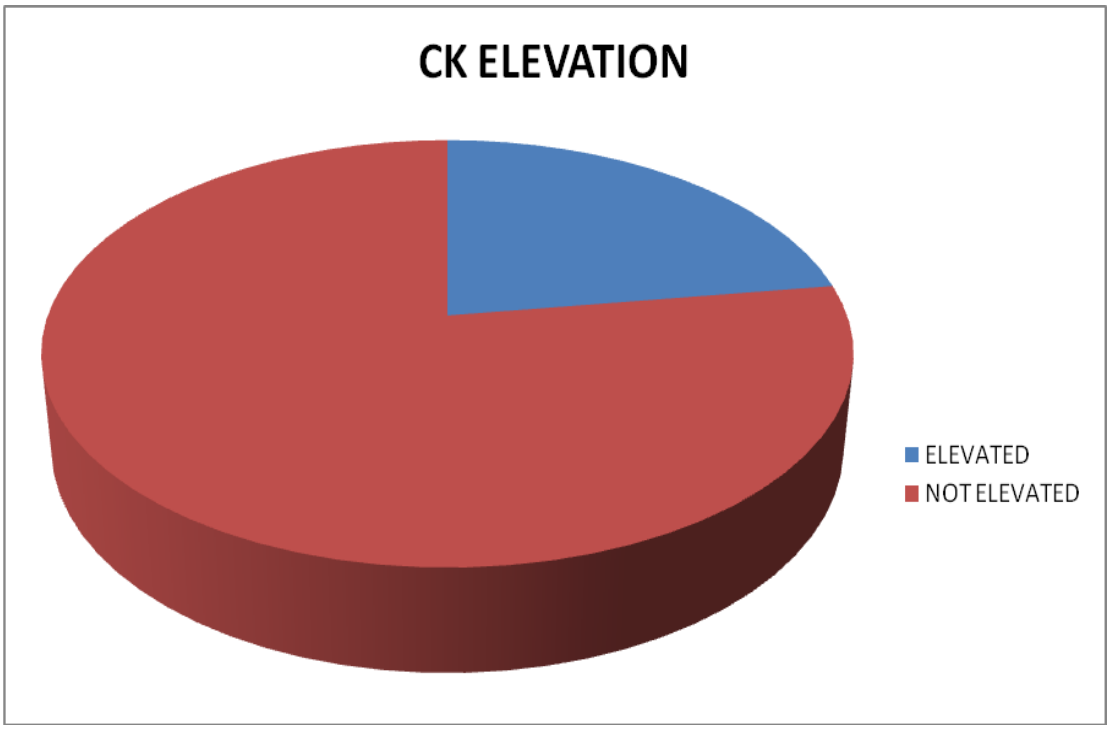



**FIG : 14**

CK-MB IN VARIOUS AGE GROUPS

**TABLE 4**

MORE HIGH AT THE UPPER LIMIT OF NORMAL IN AGE> 60

| 30 | | | | | 95% Confidence Interval for Mean | | | |
|---|---|---|---|---|---|---|---|---|
| | **N** | **Mean** | **Std. Deviation** | **Std. Error** | **Lower Bound** | **Upper Bound** | **Minimum** | **Maximum** |
| AGE<40 | 10 | 3.693000 | 2.4649816 | .7794956 | 1.929658 | 5.456342 | .0600 | 9.0000 |
| AGE 40-60 | 31 | 3.539677 | 2.1680459 | .3893925 | 2.744432 | 4.334923 | .0800 | 9.0000 |
| AGE>60 | 34 | 4.921176 | 2.8383262 | .4867689 | 3.930838 | 5.911515 | .4600 | 10.8000 |
| Total | 75 | 4.186400 | 2.5871453 | .2987378 | 3.591152 | 4.781648 | .0600 | 10.8000 |

**ANOVA**

## TABLE 5

**CK-MB**

| | Sum of Squares | Df | Mean Square | F | Sig. |
|---|---|---|---|---|---|
| Between Groups | 33.757 | 2 | 16.878 | 2.633 | .079 |
| Within Groups | 461.549 | 72 | 6.410 | | |
| Total | 495.306 | 74 | | | |

THIS TABLE SHOWS A MARGINAL SIGNIFICANCE OF TROPONIN ELEVATION IN AGE> 60 YRS

## TABLE 6

**TROPONIN I**

| 31 | | | | | 95% Confidence Interval for Mean | | | |
|---|---|---|---|---|---|---|---|---|
| | N | Mean | Std. Deviation | Std. Error | Lower Bound | Upper Bound | Minimum | Maximum |
| AGE<40 | 10 | .494000 | .3242838 | .1025475 | .262021 | .725979 | .0400 | .9200 |
| AGE 40-60 | 31 | .536774 | .4165284 | .0748107 | .383990 | .689558 | .0200 | 1.4600 |
| AGE>60 | 34 | .705588 | .5956468 | .1021526 | .497757 | .913419 | .0200 | 2.8000 |
| Total | 75 | .607600 | .4996021 | .0576891 | .492652 | .722548 | .0200 | 2.8000 |

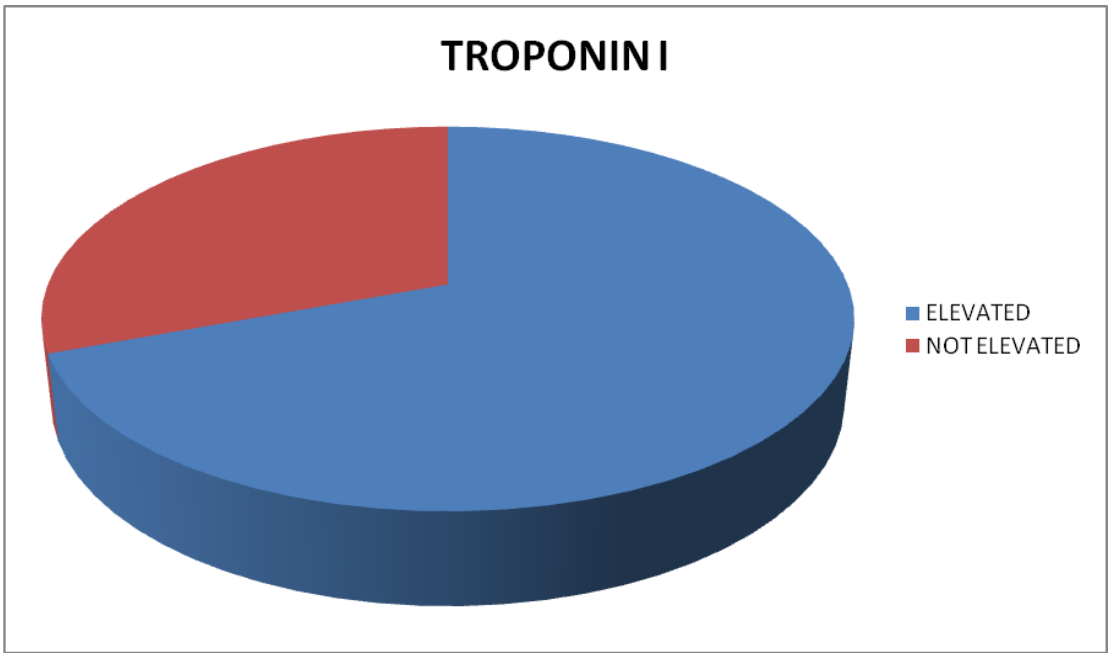



**FIG : 15**

**ANOVA**

**TABLE 7**

**TROPONIN I**

| | Sum of Squares | Df | Mean Square | F | Sig. |
|---|---|---|---|---|---|
| Between Groups | .611 | 2 | .306 | 1.232 | .298 |
| Within Groups | 17.860 | 72 | .248 | | |
| Total | 18.471 | 74 | | | |

THERE IS NO SIGNIFICANT ELEVATION IN THE VARIOUS AGE GROUPS

## MULTIPLE COMPARISONS

# TABLE 8

**Tn-I**

| 32 | (J) AGEGROUP | Mean Difference (I-J) | Std. Error | Sig. | 95% Confidence Interval | |
|---|---|---|---|---|---|---|
| | | | | | Lower Bound | Upper Bound |
| 1 | 40-60YR | -.0427742 | .1811257 | .814 | -.403842 | .318293 |
| | >60 YRS | -.2115882 | .1791661 | .242 | -.568749 | .145573 |
| 2 | 20-40 | .0427742 | .1811257 | .814 | -.318293 | .403842 |
| | >60 YRS | -.1688140 | .1236817 | .177 | -.415369 | .077741 |
| 3 | 20-40YR | .2115882 | .1791661 | .242 | -.145573 | .568749 |
| | 40-60 YR | .1688140 | .1236817 | .177 | -.077741 | .415369 |

# TABLE - 9

## Descriptives

| | | | | | | 95% Confidence Interval for Mean | |
|---|---|---|---|---|---|---|---|
| | | N | Mean | Std. Deviation | Std. Error | Lower Bound | Upper Bound |
| CK-MB | >60 | 66 | 4.385303 | 2.6352511 | .3243770 | 3.737477 | 5.033129 |
| | 40-60 | 7 | 3.208571 | 1.4679172 | .5548206 | 1.850974 | 4.566168 |
| | 20-40 | 1 | .060000 | . | . | . | . |
| | <20 | 1 | 2.030000 | . | . | . | . |
| | Total | 75 | 4.186400 | 2.5871453 | .2987378 | 3.591152 | 4.781648 |
| Tn-i | >60 | 66 | .626515 | .5218915 | .0642404 | .498218 | .754812 |
| | 40-60 | 7 | .528571 | .2451821 | .0926701 | .301816 | .755327 |
| | 20-40 | 1 | .040000 | . | . | . | . |
| | <20 | 1 | .480000 | . | . | . | . |
| | Total | 75 | .607600 | .4996021 | .0576891 | .492652 | .722548 |

**TABLE - 10**

**Descriptives**

| | | | |
|---|---|---|---|
| | | **Minimum** | **Maximum** |
| CK-MB | >60 | .0800 | 10.8000 |
| | 40-60 | .4600 | 4.8000 |
| | 20-40 | .0600 | .0600 |
| | <20 | 2.0300 | 2.0300 |
| | Total | .0600 | 10.8000 |
| Tn-i | >60 | .0200 | 2.8000 |
| | 40-60 | .0800 | .8000 |
| | 20-40 | .0400 | .0400 |
| | <20 | .4800 | .4800 |
| | Total | .0200 | 2.8000 |

**ANOVA**

## TABLE 11

| | | Sum of Squares | Df | Mean Square | F | Sig. |
|---|---|---|---|---|---|---|
| CK-MB | Between Groups | 30.981 | 3 | 10.327 | 1.579 | .202 |
| | Within Groups | 464.324 | 71 | 6.540 | | |
| | Total | 495.306 | 74 | | | |
| Tn-i | Between Groups | .406 | 3 | .135 | .532 | .662 |
| | Within Groups | 18.065 | 71 | .254 | | |
| | Total | 18.471 | 74 | | | |

FROM THE ABOVE TABLE, THE INFERENCE IS AGE HAVE NO IMPACT ON THE ELEVATED ENZYMES IN SVT PATIENTS

**Correlations**

**TABLE 12**

**CORRELATION OF NO OF EPISODE WITH ENZYME ELEVATION**

| | | EPISODE | CK-MB | Tn-i |
|---|---|---|---|---|
| EPISODE | Pearson Correlation | 1 | -.228* | -.113 |
| | Sig. (2-tailed) | | .049 | .333 |
| | N | 75 | 75 | 75 |
| CK-MB | Pearson Correlation | -.228* | 1 | .340** |
| | Sig. (2-tailed) | .049 | | .003 |
| | N | 75 | 75 | 75 |
| Tn-i | Pearson Correlation | -.113 | .340** | 1 |
| | Sig. (2-tailed) | .333 | .003 | |
| | N | 75 | 75 | 75 |

FROM THE ABOVE TABLE THERE IS SIGNIFICANT PROBABILITY BETWEEN NO OF EPISODES OF SVT WITH CK(MB) . NOSIGNIFICANT ASSOCIATION BETWEEN TROPONIN AND THE NO OF EPISODE

**CORRELATION OF NO OF EPISODE WITH ENZYME ELEVATION**

| | | EPISODE | CK-MB | Tn-i |
|---|---|---|---|---|
| EPISODE | Pearson Correlation | 1 | -.228* | -.113 |
| | Sig. (2-tailed) | | .049 | .333 |
| | N | 75 | 75 | 75 |
| CK-MB | Pearson Correlation | -.228* | 1 | .340** |
| | Sig. (2-tailed) | .049 | | .003 |
| | N | 75 | 75 | 75 |
| Tn-i | Pearson Correlation | -.113 | .340** | 1 |
| | Sig. (2-tailed) | .333 | .003 | |
| | N | 75 | 75 | 75 |

FROM THE ABOVE TABLE THERE IS SIGNIFICANT PROBABILITY BETWEEN NO OF EPISODES OF SVT WITH CK(MB) . NOSIGNIFICANT ASSOCIATION BETWEEN TROPONIN AND THE NO OF EPISODE

**SEX AND AGE GROUP**

**TABLE 13**

| | | | AGEGROUP | | | |
|---|---|---|---|---|---|---|
| | | | **<40** | **40-60** | **>60** | **Total** |
| SEX | 1 | Count | 5 | 24 | 19 | 48 |
| | | % within AGEGROUP | 50.0% | 77.4% | 55.9% | 64.0% |
| | | % of Total | 6.7% | 32.0% | 25.3% | 64.0% |
| | 2 | Count | 5 | 7 | 15 | 27 |
| | | % within AGEGROUP | 50.0% | 22.6% | 44.1% | 36.0% |
| | | % of Total | 6.7% | 9.3% | 20.0% | 36.0% |
| | Total | Count | 10 | 31 | 34 | 75 |
| | | % within AGEGROUP | 100.0% | 100.0% | 100.0% | 100.0% |
| | | % of Total | 13.3% | 41.3% | 45.3% | 100.0% |

Chi-Square Tests **TABLE 14**

| | **Value** | **Df** | **Asymp. Sig. (2-sided)** |
|---|---|---|---|
| Pearson Chi-Square | 4.246a | 2 | .120 |
| Likelihood Ratio | 4.370 | 2 | .113 |
| Linear-by-Linear Association | .218 | 1 | .641 |
| N of Valid Cases | 75 | | |

FROM THE CORRELATION, THERE IS NO SIGNIFICANT PROBABILITY WITH AGE OF INCIDENCE OF SVT WITH SEX. BOTH ARE EQUALLY AFFECTED

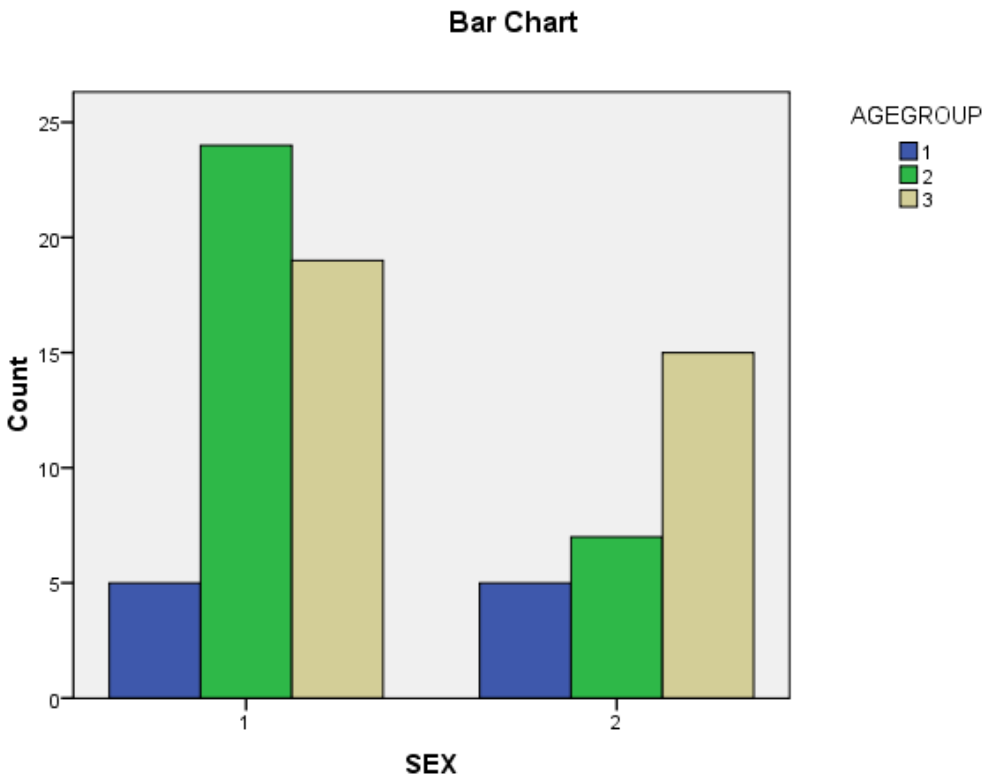



**FIG : 16**

**EPISODE * AGEGROUP**

**TABLE 15**

| | | | AGEGROUP | | | |
|---|---|---|---|---|---|---|
| | | | **<40** | **40-60** | **>60** | **Total** |
| EPISODE | 1 | Count | 8 | 28 | 30 | 66 |
| | | % within AGEGROUP | 80.0% | 90.3% | 88.2% | 88.0% |
| | | % of Total | 10.7% | 37.3% | 40.0% | 88.0% |
| | 2 | Count | 0 | 3 | 4 | 7 |
| | | % within AGEGROUP | .0% | 9.7% | 11.8% | 9.3% |
| | | % of Total | .0% | 4.0% | 5.3% | 9.3% |
| | 3 | Count | 1 | 0 | 0 | 1 |
| | | % within AGEGROUP | 10.0% | .0% | .0% | 1.3% |
| | | % of Total | 1.3% | .0% | .0% | 1.3% |
| | 4 | Count | 1 | 0 | 0 | 1 |
| | | % within AGEGROUP | 10.0% | .0% | .0% | 1.3% |
| | | % of Total | 1.3% | .0% | .0% | 1.3% |
| | Total | Count | 10 | 31 | 34 | 75 |
| | | % within AGEGROUP | 100.0% | 100.0% | 100.0% | 100.0% |
| | | % of Total | 13.3% | 41.3% | 45.3% | 100.0% |

Chi-Square Tests

**TABLE 16**

| | **Value** | **df** | **Asymp. Sig. (2-sided)** |
|---|---|---|---|
| Pearson Chi-Square | 14.245a | 6 | .027 |
| Likelihood Ratio | 10.223 | 6 | .116 |
| Linear-by-Linear Association | 2.640 | 1 | .104 |
| N of Valid Cases | 75 | | |

FROM ABOVE TABLE COMPARING VARIOUS AGE GROUP WITH NO OF EPISODES, IT SEEMS THAT THERE IS SIGNIFICANT PROBABILITY

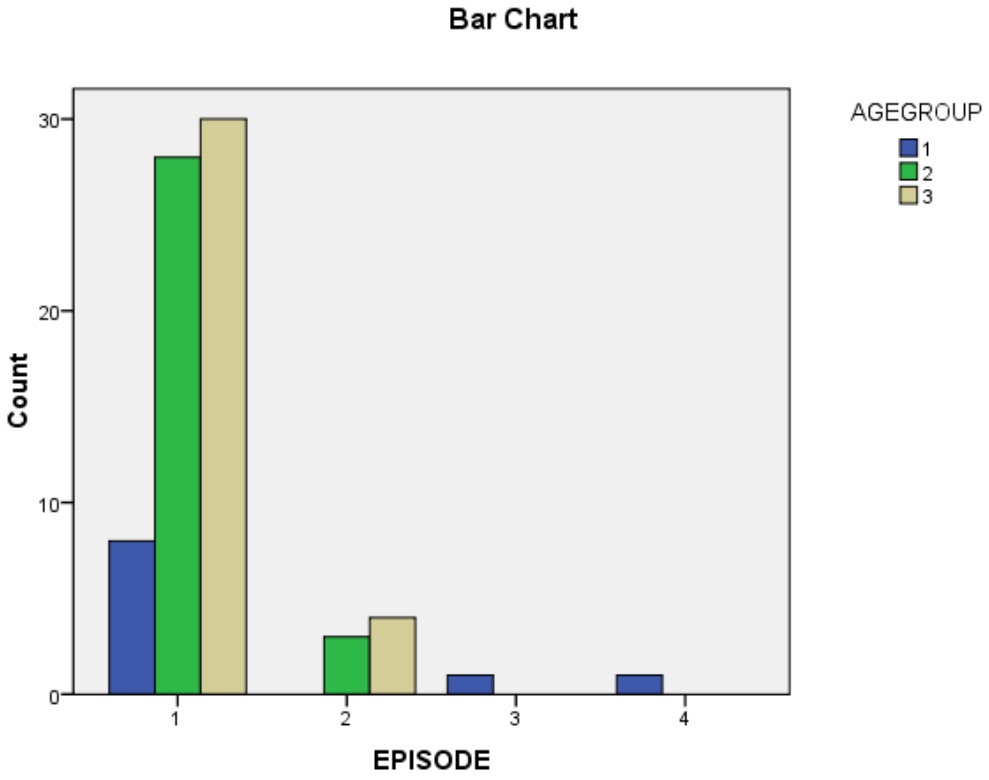



**FIG : 17**

**SMOKING * AGEGROUP**

**TABLE 17**

| | | | AGEGROUP | | | |
|---|---|---|---|---|---|---|
| | | | <40 | 40-60 | >60 | Total |
| SMOKING | NON SMOKER | Count | 6 | 14 | 24 | 44 |
| | | % within AGEGROUP | 60.0% | 45.2% | 70.6% | 58.7% |
| | | % of Total | 8.0% | 18.7% | 32.0% | 58.7% |
| | SMOKER | Count | 4 | 17 | 10 | 31 |
| | | % within AGEGROUP | 40.0% | 54.8% | 29.4% | 41.3% |
| | | % of Total | 5.3% | 22.7% | 13.3% | 41.3% |
| | Total | Count | 10 | 31 | 34 | 75 |
| | | % within AGEGROUP | 100.0% | 100.0% | 100.0% | 100.0% |
| | | % of Total | 13.3% | 41.3% | 45.3% | 100.0% |

Chi-Square Tests

**TABLE 18**

| | Value | Df | Asymp. Sig. (2-sided) |
|---|---|---|---|
| Pearson Chi-Square | 4.332a | 2 | .115 |
| Likelihood Ratio | 4.369 | 2 | .113 |
| Linear-by-Linear Association | 1.721 | 1 | .190 |
| N of Valid Cases | 75 | | |

THERE IS NOSIGNIFICANT DIFFERENCE WITH OCCURRENCE OF SVT AND SMOKING

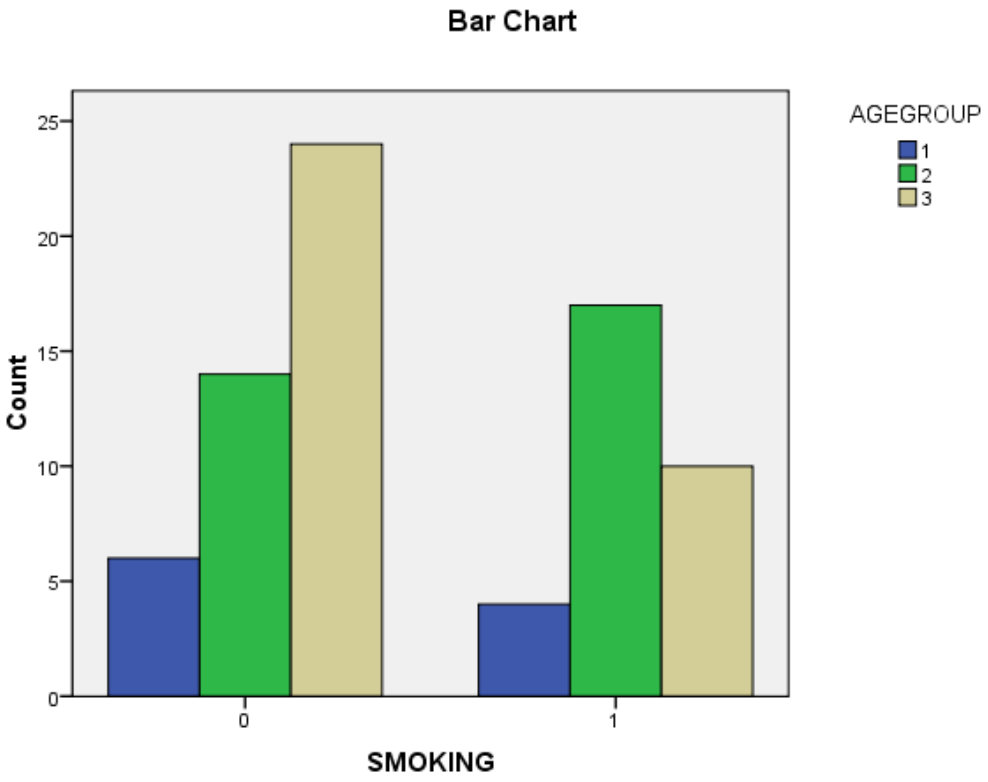



FIG : 18

**ALCOHOL * AGEGROUP**

**TABLE 19**

| | | | AGEGROUP | | | |
|---|---|---|---|---|---|---|
| | | | **<40** | **40-60** | **>60** | **Total** |
| ALCOHOL | NONALCOHOLIC | Count | 7 | 17 | 27 | 51 |
| | | % within AGEGROUP | 70.0% | 54.8% | 79.4% | 68.0% |
| | | % of Total | 9.3% | 22.7% | 36.0% | 68.0% |
| | ALCOHOLIC | Count | 3 | 14 | 7 | 24 |
| | | % within AGEGROUP | 30.0% | 45.2% | 20.6% | 32.0% |
| | | % of Total | 4.0% | 18.7% | 9.3% | 32.0% |
| | Total | Count | 10 | 31 | 34 | 75 |
| | | % within AGEGROUP | 100.0% | 100.0% | 100.0% | 100.0% |
| | | % of Total | 13.3% | 41.3% | 45.3% | 100.0% |

Chi-Square Tests

**TABLE 20**

| | **Value** | **Df** | **Asymp. Sig. (2-sided)** |
|---|---|---|---|
| Pearson Chi-Square | 4.521a | 2 | .104 |
| Likelihood Ratio | 4.554 | 2 | .103 |
| Linear-by-Linear Association | 1.691 | 1 | .194 |
| N of Valid Cases | 75 | | |

THE INFERENCE FROM ABOVE TABLE IS THERE IS NO SIGNIFICANT DIFFERENCE BETWEEN BOTH ALCOHOLIC AND NON ALCOHOLIC WITH OCCURRENCE OF SVT

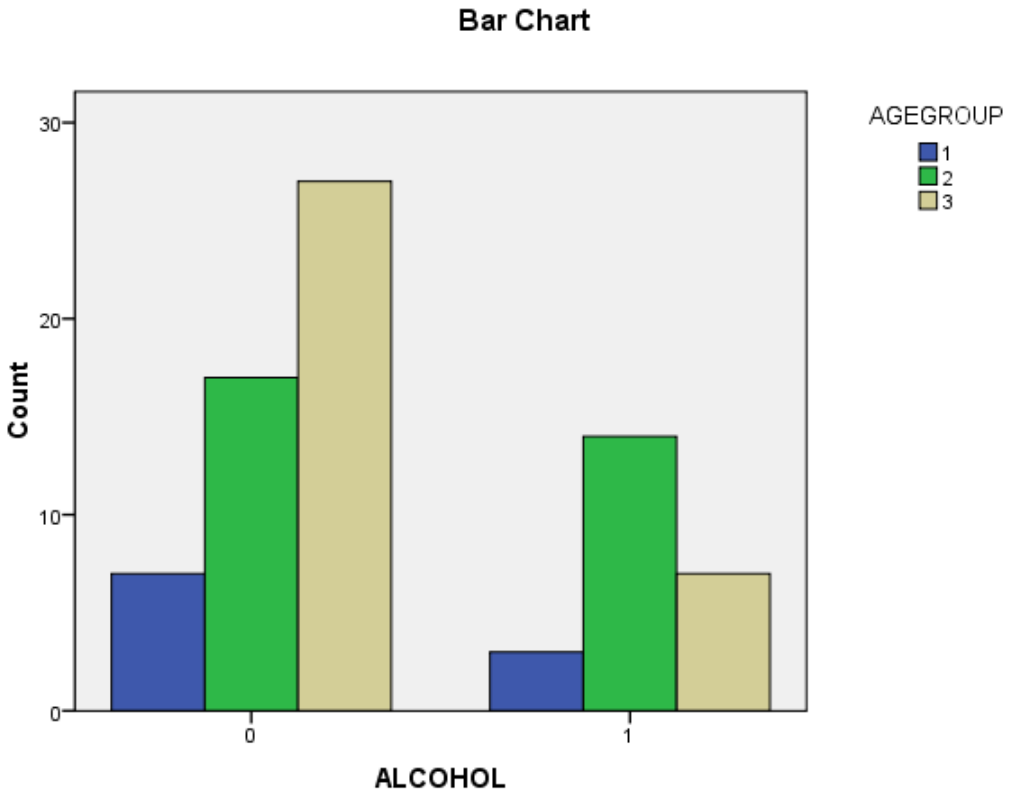



**FIG : 19**

DIABETES * AGEGROUP

**TABLE 21**

| | | | AGEGROUP | | | |
|---|---|---|---|---|---|---|
| | | | <40 | 40-60 | >60 | Total |
| DIABETES | NON DIABETIC | Count | 9 | 16 | 17 | 42 |
| | | % within AGEGROUP | 90.0% | 51.6% | 50.0% | 56.0% |
| | | % of Total | 12.0% | 21.3% | 22.7% | 56.0% |
| | DIABETIC | Count | 1 | 15 | 17 | 33 |
| | | % within AGEGROUP | 10.0% | 48.4% | 50.0% | 44.0% |
| | | % of Total | 1.3% | 20.0% | 22.7% | 44.0% |
| | Total | Count | 10 | 31 | 34 | 75 |
| | | % within AGEGROUP | 100.0% | 100.0% | 100.0% | 100.0% |
| | | % of Total | 13.3% | 41.3% | 45.3% | 100.0% |

Chi-Square Tests

**TABLE 22**

| | **Value** | **df** | **Asymp. Sig. (2-sided)** |
|---|---|---|---|
| Pearson Chi-Square | 5.430a | 2 | .066 |
| Likelihood Ratio | 6.311 | 2 | .043 |
| Linear-by-Linear Association | 3.263 | 1 | .071 |
| N of Valid Cases | 75 | | |

THERE IS SIGNIFICANT DIFFERENCE WITH OCCURRENCE OF SVT IN DIABETIC AND NON DIABETIC

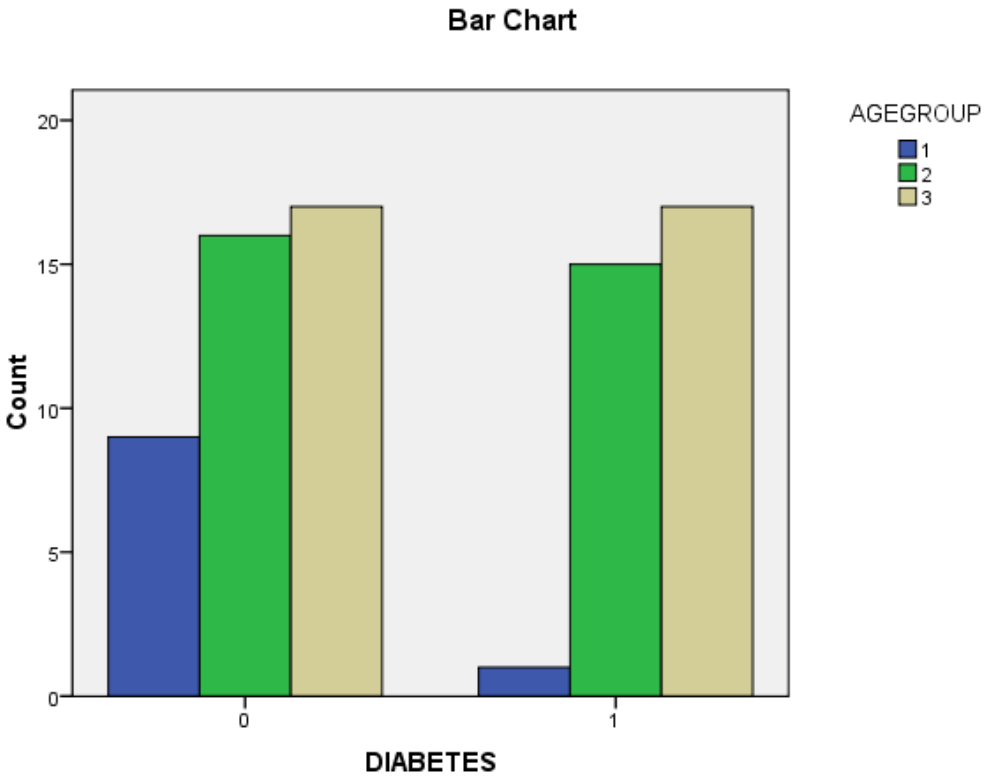



**FIG - 20**

**HYPERTENSION * AGEGROUP**

**TABLE 23**

| | | | AGEGROUP | | | |
|---|---|---|---|---|---|---|
| | | | <40 | 40-60 | >60 | Total |
| HYPERTENSION | NO | Count | 8 | 17 | 15 | 40 |
| | | % within AGEGROUP | 80.0% | 54.8% | 44.1% | 53.3% |
| | | % of Total | 10.7% | 22.7% | 20.0% | 53.3% |
| | YES | Count | 2 | 14 | 19 | 35 |
| | | % within AGEGROUP | 20.0% | 45.2% | 55.9% | 46.7% |
| | | % of Total | 2.7% | 18.7% | 25.3% | 46.7% |
| | Total | Count | 10 | 31 | 34 | 75 |
| | | % within AGEGROUP | 100.0% | 100.0% | 100.0% | 100.0% |
| | | % of Total | 13.3% | 41.3% | 45.3% | 100.0% |

Chi-Square Tests

**TABLE 24**

| | **Value** | **df** | **Asymp. Sig. (2-sided)** |
|---|---|---|---|
| Pearson Chi-Square | 4.046a | 2 | .132 |
| Likelihood Ratio | 4.284 | 2 | .117 |
| Linear-by-Linear Association | 3.672 | 1 | .055 |
| N of Valid Cases | 75 | | |

FROM THE ABOVE TABLE THERE IS NO DIFFERENCE IN OCCURRENCE OF SVT IN HYPERTENSIVES AND NON HYPERTENSIVES

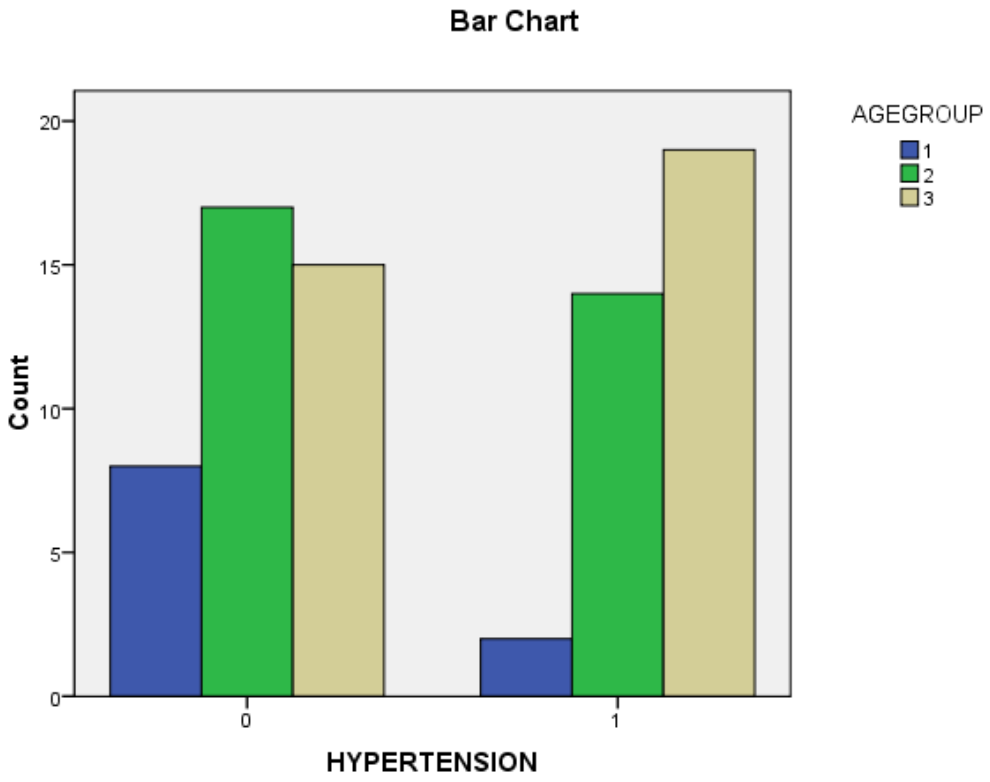



FIG 21

ECG * AGEGROUP

**TABLE 25**

| | | | AGEGROUP | | | |
|---|---|---|---|---|---|---|
| | | | **<40** | **40-60** | **>60** | **Total** |
| ECG | AVNRT | Count | 8 | 30 | 32 | 70 |
| | | % within AGEGROUP | 80.0% | 96.8% | 94.1% | 93.3% |
| | | % of Total | 10.7% | 40.0% | 42.7% | 93.3% |
| | AVRT | Count | 2 | 1 | 2 | 5 |
| | | % within AGEGROUP | 20.0% | 3.2% | 5.9% | 6.7% |
| | | % of Total | 2.7% | 1.3% | 2.7% | 6.7% |
| | Total | Count | 10 | 31 | 34 | 75 |
| | | % within AGEGROUP | 100.0% | 100.0% | 100.0% | 100.0% |
| | | % of Total | 13.3% | 41.3% | 45.3% | 100.0% |

Chi-Square Tests

**TABLE 26**

| | **Value** | **Df** | **Asymp. Sig. (2-sided)** |
|---|---|---|---|
| Pearson Chi-Square | 3.481a | 2 | .175 |
| Likelihood Ratio | 2.683 | 2 | .261 |
| N of Valid Cases | 75 | | |

THERE IS A PROPORTIONATE INCIDENCE IN OCCURRENCE OF SVT IN 3 DIFFERENT AGE GROUPS but no significant correlation between age and type of SVT

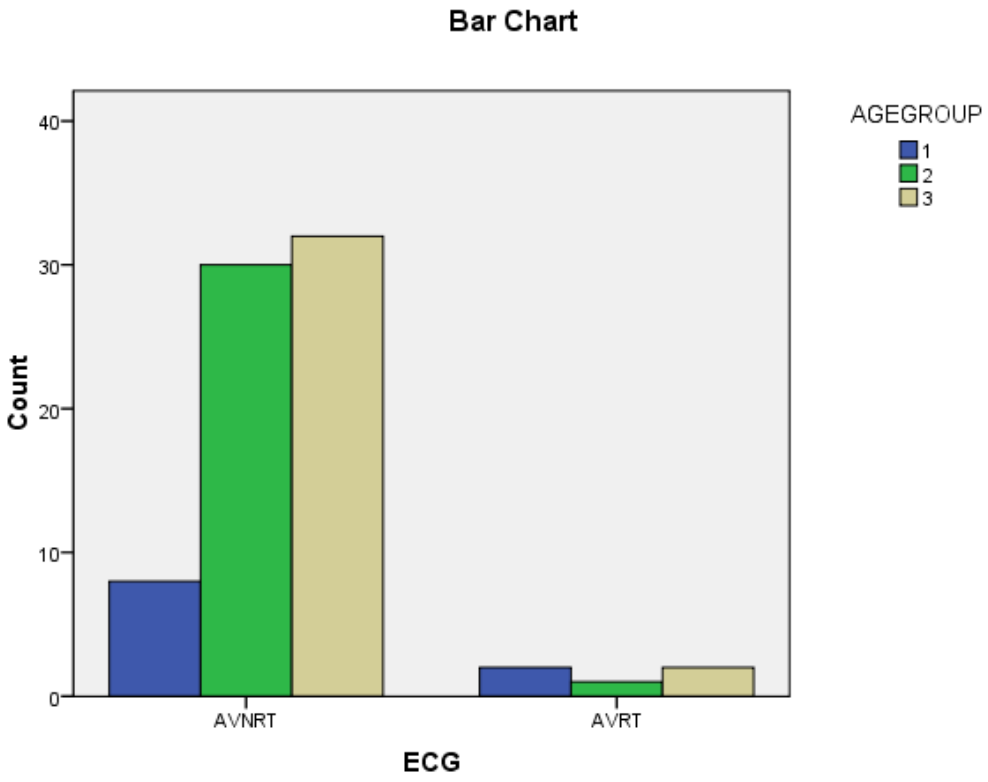



**FIG - 22**

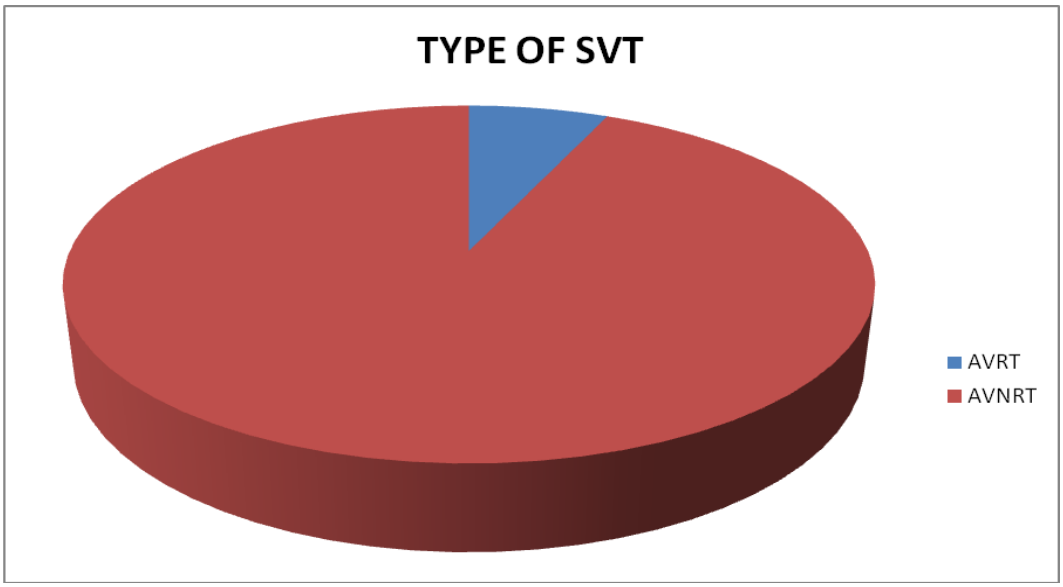



**FIG : 23**

## ECHO * AGEGROUP

## TABLE 27

| | | | AGEGROUP | | | |
|---|---|---|---|---|---|---|
| | | | <40 | 40-60 | >60 | Total |
| ECHO | N STUDY | Count | 6 | 23 | 19 | 48 |
| | | % within AGEGROUP | 60.0% | 74.2% | 55.9% | 64.0% |
| | | % of Total | 8.0% | 30.7% | 25.3% | 64.0% |
| | AV SCLEROSIS | Count | 0 | 1 | 1 | 2 |
| | | % within AGEGROUP | .0% | 3.2% | 2.9% | 2.7% |
| | | % of Total | .0% | 1.3% | 1.3% | 2.7% |
| | ATHELETE' S HEART | Count | 1 | 0 | 0 | 1 |
| | | % within AGEGROUP | 10.0% | .0% | .0% | 1.3% |
| | | % of Total | 1.3% | .0% | .0% | 1.3% |
| | GRADE 1,2DD | Count | 0 | 1 | 1 | 2 |
| | | % within AGEGROUP | .0% | 3.2% | 2.9% | 2.7% |
| | | % of Total | .0% | 1.3% | 1.3% | 2.7% |
| | GRADE3, DD | Count | 0 | 5 | 10 | 15 |
| | | % within AGEGROUP | .0% | 16.1% | 29.4% | 20.0% |
| | | % of Total | .0% | 6.7% | 13.3% | 20.0% |
| | GRADE 4DD | Count | 0 | 0 | 1 | 1 |
| | | % within AGEGROUP | .0% | .0% | 2.9% | 1.3% |
| | | % of Total | .0% | .0% | 1.3% | 1.3% |
| | AORTIC STENOSIS | Count | 0 | 0 | 1 | 1 |
| | | % within AGEGROUP | .0% | .0% | 2.9% | 1.3% |
| | | % of Total | .0% | .0% | 1.3% | 1.3% |
| | MITRL VALVE PROLAPSE | Count | 1 | 1 | 0 | 2 |
| | | % within AGEGROUP | 10.0% | 3.2% | .0% | 2.7% |
| | | % of Total | 1.3% | 1.3% | .0% | 2.7% |
| | WPW | Count | 2 | 0 | 1 | 3 |
| | | % within AGEGROUP | 20.0% | .0% | 2.9% | 4.0% |
| | | % of Total | 2.7% | .0% | 1.3% | 4.0% |
| | Total | Count | 10 | 31 | 34 | 75 |
| | | % within AGEGROUP | 100.0% | 100.0% | 100.0% | 100.0% |
| | | % of Total | 13.3% | 41.3% | 45.3% | 100.0% |

**CHISQUARETESTS**

**TABLE 28**

| | Value | df | Asymp. Sig. (2-sided) |
|---|---|---|---|
| Pearson Chi-Square | 24.848a | 16 | .073 |
| Likelihood Ratio | 23.627 | 16 | .098 |
| Linear-by-Linear Association | .000 | 1 | .996 |
| N of Valid Cases | 75 | | |

There is a significant correlation between occurrence of SVT with various echo findings.

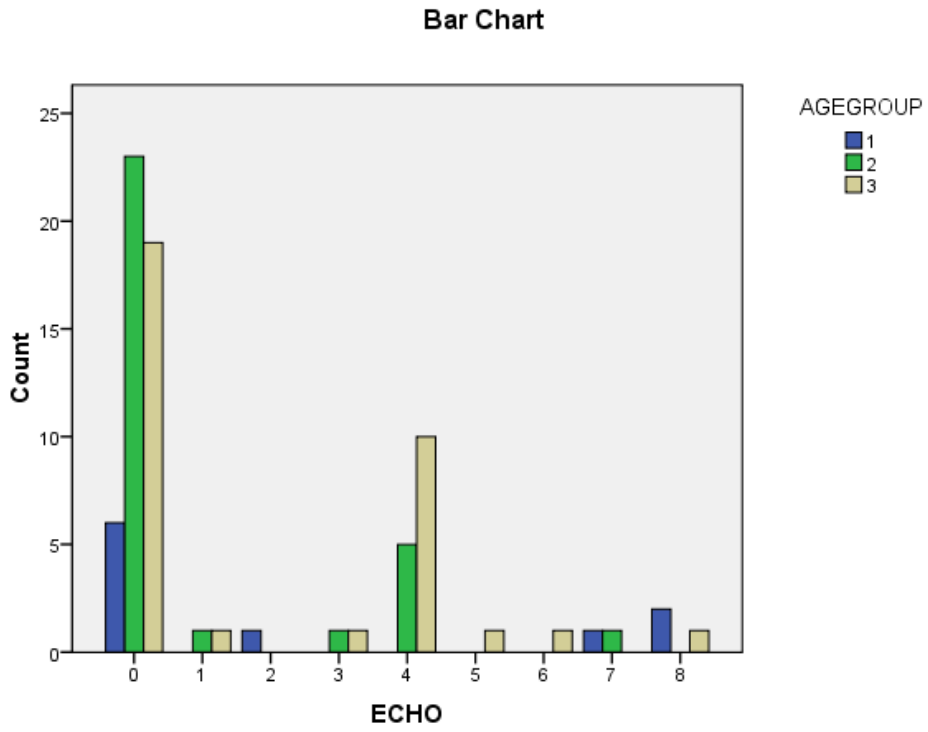



**FIG : 24**

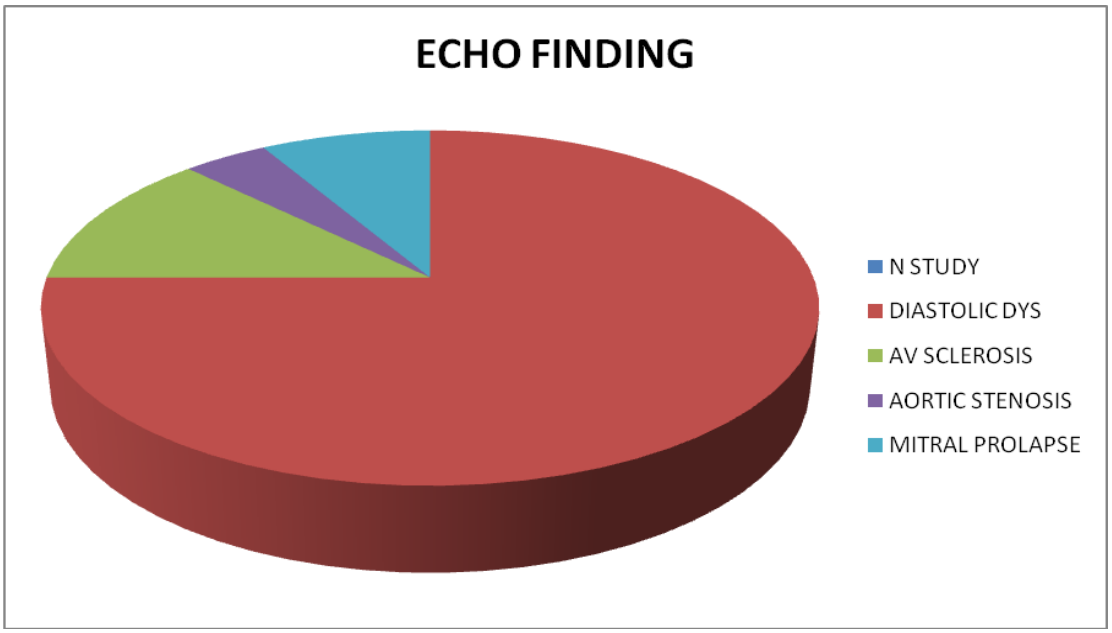

ECHO FINDING
N STUDY
DIASTOLIC DYS
AV SCLEROSIS
AORTIC STENOSIS
MITRAL PROLAPSE

**FIG 25**

LVH * AGEGROUP

**TABLE 29**

| | | | AGEGROUP | | | |
|---|---|---|---|---|---|---|
| | | | <40 | 40-60 | >60 | Total |
| LVH | NO | Count | 9 | 23 | 16 | 48 |
| | | % within AGEGROUP | 90.0% | 74.2% | 47.1% | 64.0% |
| | | % of Total | 12.0% | 30.7% | 21.3% | 64.0% |
| | YES | Count | 1 | 8 | 18 | 27 |
| | | % within AGEGROUP | 10.0% | 25.8% | 52.9% | 36.0% |
| | | % of Total | 1.3% | 10.7% | 24.0% | 36.0% |
| | Total | Count | 10 | 31 | 34 | 75 |
| | | % within AGEGROUP | 100.0% | 100.0% | 100.0% | 100.0% |
| | | % of Total | 13.3% | 41.3% | 45.3% | 100.0% |

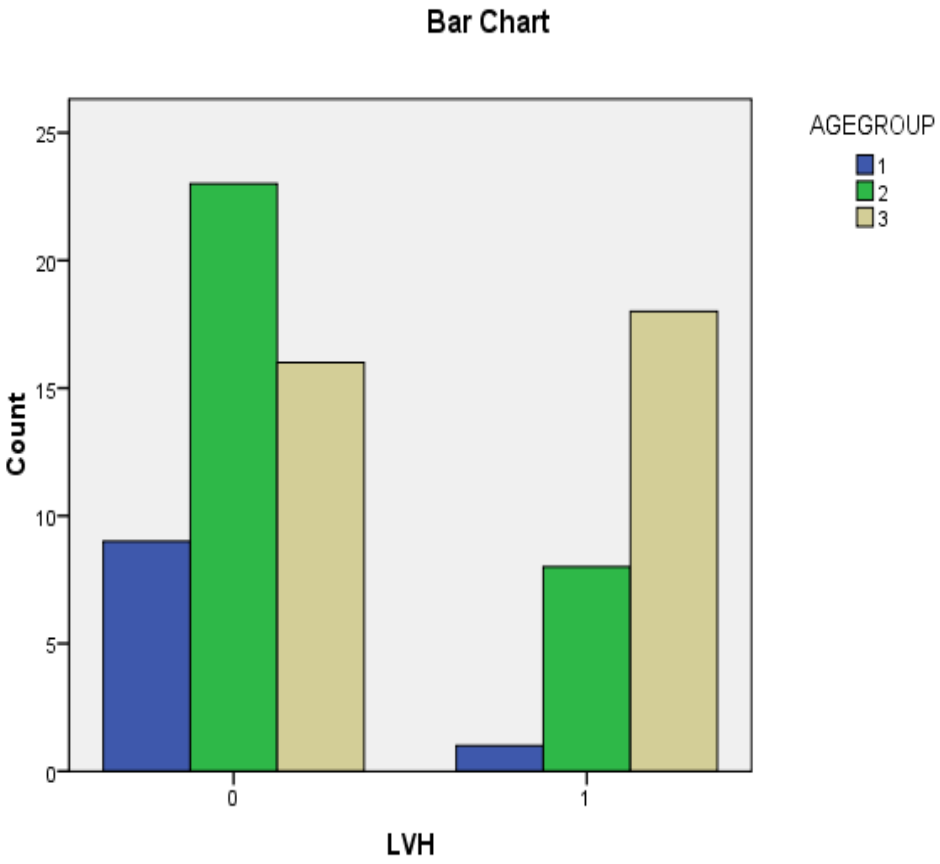



**FIG : 26**

Chi-Square Tests

**TABLE 30**

| | Value | df | Asymp. Sig. (2-sided) |
|---|---|---|---|
| Pearson Chi-Square | 8.567a | 2 | .014 |
| Likelihood Ratio | 9.091 | 2 | .011 |
| Linear-by-Linear Association | 8.241 | 1 | .004 |
| N of Valid Cases | 75 | | |

THE INFERENCE FROM ABOVE TABLE THERE IS SIGNIFICANCE IN OCURRENCE OF SVT WITH PRESENCE OF LVH

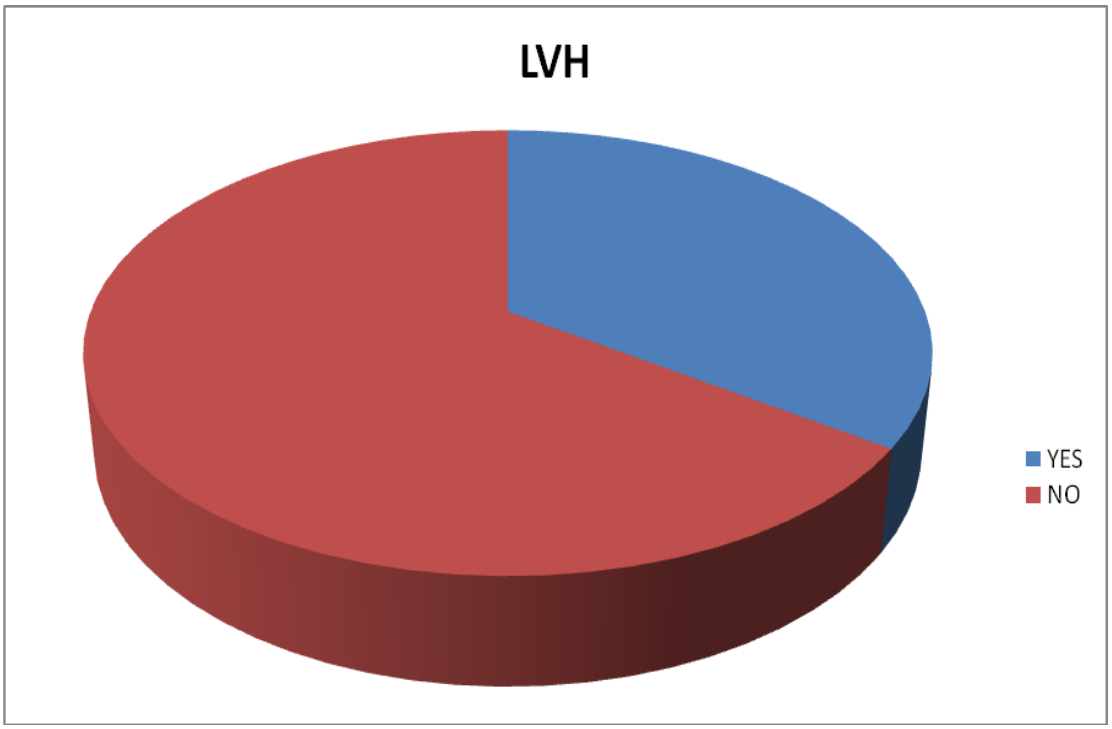



**FIG : 27**
**HEMOGLOBIN**

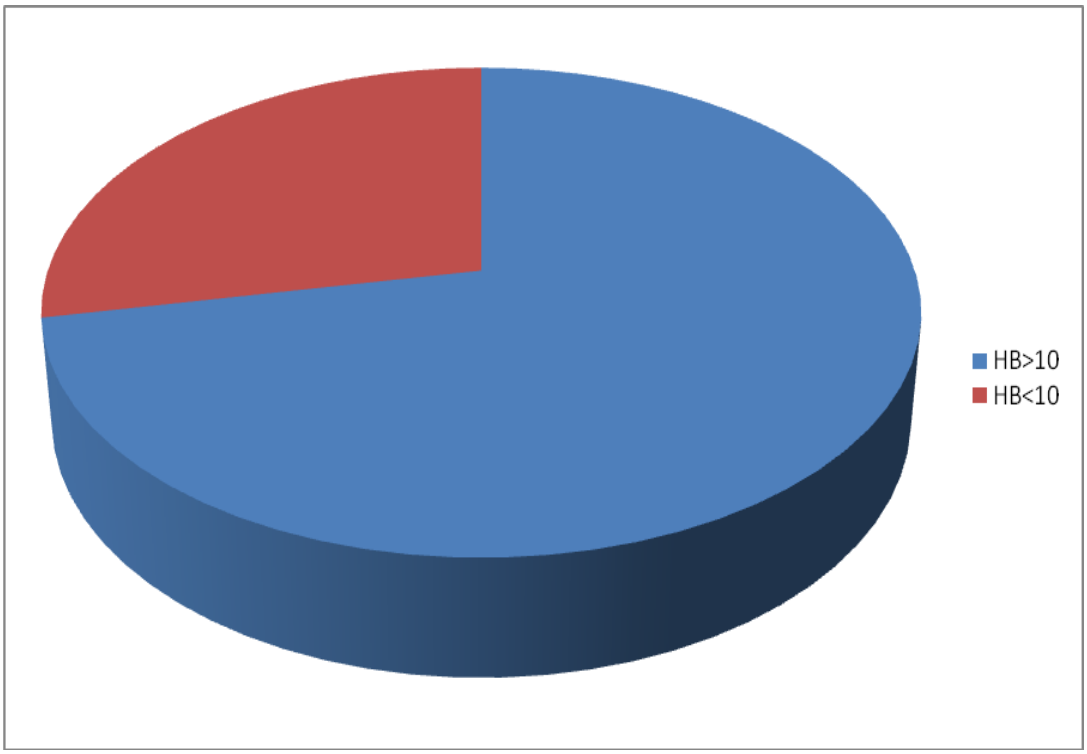



**FIG : 28**

## INFERENCES FROM THE STUDY

By pearson correlation analysis

1. There is no correlation between various age group, occurrence of SVT.

2. There is significant  no correlation between occurrence of SVT with sex.

3. There is no significant correlation between occurrence of SVT with alcohol and smoking.

4. There is a significant correlation of SVT occurrence and diabetes but there is  no correlation with hypertension there is significant correlation between weight and troponin  I which is directly proportional.

5. There is a direct correlation between the no of episodes with age more than 60 affected.

6. There is direct correlation of left ventricular hypertrophy in ECHO with incidence of SVT.

7. There is also a direct correlation between weight and CK(MB) in this study.

8. There is also a direct correlation between weight and CK(MB) in this study.

9. There is correlation between hemoglobin and CK(MB) levels.

10. There is correlation between age and CK (MB) levels.

11. Systolic BP is in correlation with CK(MB) and troponin I. BOTH are directly proportional

12. There is a significant correlation with diastolic BP and troponin I levels.

# DISCUSSION AND INTERPRETATION

In this observational study I have studied I first calculated the prevalence of SVT in non CAD and non valvular heart disease patients.

The prevalence is about 80 cases within study period

All the patients underwent complete investigation and they were analysed whether there is any significant elevation of both creatinine kinase and troponin I levels in this patient and whether there is any association between various risk factors and the level of enzymes

* The mean age group is 57 years

* The mean elevation of CK(MB) is 4.18ng/ml

* The mean elevation of troponin I is 0.60 ng/ml

* The cardiac enzyme elevation is directly proportional to both systolic and diastolisc BP

* The creatinine kinase elevation increases with increase in age

* The level of hemoglobin is indirectly proportional to creatinine kinase level. With anemia there will be increasing level of (CK-MB) levels. This anemia is more common in old age and common in females compared to males

* Those patients who are hypertensive have higher incidence of SVT compared to non hypertensive patients

* There is a significant difference in occurrence of SVT with Diabetics and Non diabetics

* There is no significant difference in both incidence of SVT and enzyme variation in smokers and non smokers

* There is no significant difference in both incidence of SVT and enzyme variation in alcoholics and non alcoholics,but alcoholics have recurrent episode of SVT

* The SVT is more common in obese patients with mean weight of above 70 kg

* With increasing incidence of obesity, there is increased prevalence of both hypertension and obesity

* The presence of left ventricular hypertrophy and diastolic dysfunction in ECHO which represents the severe form of hypertension is more common in older age compared to middle age and they have direct correlation with occurrence of SVT

* There is no significant correlation between the elevated troponin I with presence of left ventricular hypertrophy in adults.

* The most commonest form of SVT is AVNRT and the patients with WPW get recurrent episodes of AVRT

* Even mitral valve prolapse patients can have sudden incidence of SVT

# CONCLUSION

Troponin levels are essential for the diagnosis of myocardial infarction. But it was not pathognomic for ACS[61]. It was found recently that it was elevated in some severe acute diseases like acute pulmonary thrombo embolism, stroke, pericarditis, ESRD, most importantly sepsis[62]. As a result the elevated troponin levels should be interpreted in text of clinical finding. Sometimes these troponin elevation leads to misdiagnosis and can cause unnecessary tests and treatments to be ordered, leading to delay in proper diagnosis and treatment[63].

In a patient with SVT, the use of troponin testing would best be performed selectively based on presenting symptoms and risk factors for acute coronary syndrome[64]. In the absence of clinical coronary artery disease, troponin levels may point to minor myocardial injury[65], as shown by the cases of supraventricular tachyarrythmia. Apart from this, the elevated troponins in these group of patients indicates worser prognosis. Follow up prospective studies are needed to prove this new concepts.

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# PROFORMA

NAME AGE SEX

DATE OF ADMISSION

COMPLAINTS

RELEVANT PRESENT HISTORY

PAST HISTORY

PREVIOUS EPISODE

DIABETES

HYPERTENSION

SMOKING

ALCOHOL

CAD

DRUGS

BP PR

GENERAL EXAMINATION

CVS EXAMINATION

INVESTIGATIONS

ECG

1.

2.

COMPLETE HEMOGRAM

RENAL FUNCTION TEST

ECHOCARDIOGRAM

CK(MB)

TROPONIN I

CARDIOLOGY OPINION

TREATMENT GIVEN

| NAME | AGE | SEX | WEIGHT | EPISODE | SMOKING | ALCOHOL | DIABETES | HYPERTENSION | HB | ECG | ECHO | CK-MB | Tn-i | sys BP | DIA BP | LVH |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| ABDUL HAMEED | 50 | M | 76 | 1 | 1 | 0 | 0 | 0 | 12 | AVNRT | N STUDY | 3.6 | 1.08 | 160 | 90 | 1 |
| AIYANAAR | 72 | M | 72 | 2 | 1 | 0 | 0 | 1 | 8.4 | AVNRT | GRADE I DD 4.80 | 4.8 | 0.56 | 150 | 90 | 1 |
| ALWYN | 56 | M | 69 | 1 | 1 | 1 | 0 | 0 | 11 | AVNRT | N STUDY | 0.08 | 0.12 | 108 | 46 | 0 |
| ANTHONIAMMAL | 56 | F | 43 | 1 | 0 | 0 | 0 | 0 | 7.8 | AVNRT | N STUDY | 8.8 | 0.8 | 110 | 80 | 0 |
| AROKYAMARY | 62 | F | 90 | 1 | 0 | 0 | 1 | 1 | 8.8 | AVNRT | N STUDY | 5.64 | 0.24 | 146 | 90 | 1 |
| ARUMUGAM | 68 | M | 68 | 1 | 1 | 0 | 0 | 0 | 8.8 | AVNRT | N STUDY | 1.8 | 0.8 | 130 | 88 | 1 |
| AZHAGARSAMY | 58 | M | 68 | 1 | 1 | 1 | 1 | 1 | 11 | AVNRT | N STUDY | 3.46 | 0.76 | 130 | 90 | 0 |
| BABUNATHAN | 43 | M | 88 | 1 | 1 | 0 | 0 | 0 | 12.8 | AVNRT | N STUDY | 2.04 | 0.02 | 140 | 88 | 1 |
| BATHRI | 88 | M | 54 | 1 | 1 | 0 | 0 | 0 | 16 | AVNRT | N STUDY | 8.88 | 0.98 | 126 | 88 | 0 |
| CHANDRASINGH | 58 | M | 88 | 1 | 1 | 0 | 0 | 0 | 11.8 | AVNRT | N STUDY | 2.84 | 0.52 | 110 | 76 | 0 |
| CHRISTOPHER | 62 | M | 68 | 2 | 0 | 0 | 1 | 0 | 12 | AVNRT | N STUDY | 2.76 | 0.42 | 150 | 70 | 0 |
| CHRISTUDAS | 48 | M | 92 | 1 | 0 | 1 | 0 | 0 | 13 | AVNRT | N STUDY | 1.04 | 0.36 | 130 | 86 | 0 |
| DANANJEYAN | 37 | M | 89 | 1 | 1 | 1 | 0 | 1 | 14 | AVNRT | N STUDY | 3.8 | 0.86 | 126 | 78 | 0 |
| DAVID | 54 | M | 78 | 1 | 1 | 1 | 0 | 0 | 9.8 | AVNRT | N STUDY | 1.08 | 1.46 | 126 | 80 | 0 |
| DENNY CLARIN | 38 | M | 83 | 1 | 1 | 1 | 0 | 0 | 13.8 | AVNRT | N STUDY | 1.26 | 0.08 | 120 | 80 | 0 |
| DEVIKA | 68 | F | 76 | 2 | 0 | 0 | 1 | 1 | 9.2 | AVNRT | N STUDY | 3.18 | 0.6 | 140 | 70 | 0 |
| DEVISHREEE | 40 | F | 58 | 1 | 1 | 0 | 1 | 0 | 8.8 | AVNRT | N STUDY | 9 | 0.6 | 110 | 80 | 0 |
| DHANASEKAR | 56 | M | 76 | 1 | 0 | 0 | 1 | 1 | 9 | AVNRT | N STUDY | 9 | 0.08 | 110 | 90 | 0 |
| DINESH | 22 | M | 56 | 1 | 0 | 0 | 0 | 0 | 12.2 | AVNRT | ATHLETE'S HEART 4 | 4 | 0.92 | 120 | 80 | 1 |
| FATHIMA | 65 | F | 49 | 1 | 0 | 0 | 1 | 1 | 13.6 | AVNRT | N STUDY | 6.8 | 0.8 | 146 | 110 | 1 |
| FEROZ KHAN | 58 | M | 70 | 1 | 0 | 1 | 1 | 0 | 11 | AVNRT | N STUDY | 3.8 | 0.98 | 112 | 88 | 0 |
| GANESH | 76 | M | 78 | 1 | 0 | 0 | 0 | 1 | 9.8 | AVNRT | N STUDY | 6.8 | 0.98 | 130 | 88 | 1 |
| GAYATHRI | 30 | F | 52 | 4 | 0 | 0 | 0 | 0 | 9.8 | AVRT | WPW | 2.03 | 0.48 | 100 | 68 | 0 |
| GEETHA | 64 | F | 46 | 1 | 0 | 0 | 1 | 1 | 9.8 | AVNRT | GRADE IDD | 2.04 | 0.14 | 130 | 78 | 0 |
| GEMINI | 64 | M | 78 | 1 | 0 | 0 | 0 | 0 | 12 | AVNRT | N STUDY | 2.08 | 1.14 | 112 | 86 | 0 |
| GOVINDAMMAL | 46 | F | 74 | 2 | 0 | 0 | 1 | 1 | 10.8 | AVRT | AV SCLEROSIS 4.8 | 4.8 | 0.78 | 110 | 90 | 0 |
| GURU | 53 | M | 90 | 1 | 1 | 1 | 1 | 0 | 11.6 | AVNRT | GRADE I DD | 1.09 | 0.32 | 110 | 88 | 0 |
| JAHARUL | 56 | M | 78 | 1 | 1 | 1 | 0 | 0 | 13 | AVNRT | N STUDY | 1.08 | 0.08 | 90 | 70 | 0 |
| JAMES EDWARD | 32 | M | 68 | 1 | 0 | 0 | 0 | 0 | 7.8 | AVNRT | MV PROLAPSE 5.04 | 5.04 | 0.2 | 130 | 72 | 0 |
| KALAISELVI | 28 | F | 76 | 1 | 0 | 0 | 0 | 0 | 12 | AVRT | WPW | 3.48 | 0.46 | 98 | 80 | 0 |
| KALAISELVI | 72 | F | 86 | 1 | 0 | 0 | 0 | 1 | 6.8 | AVNRT | ILD AS 1.04 ( | 1.04 | 0.3 | 160 | 60 | 1 |
| KARTHIKEYAN | 42 | M | 80 | 1 | 0 | 0 | 0 | 1 | 9.2 | AVNRT | N STUDY | 2.06 | 0.72 | 126 | 90 | 0 |
| KATHIRAVAN | 48 | M | 46 | 1 | 0 | 0 | 1 | 0 | 10.8 | AVNRT | MV PROLAPSE 5.04 | 6.04 | 1.06 | 110 | 80 | 0 |
| KRISHNAMOORHY | 78 | M | 88 | 1 | 0 | 0 | 0 | 0 | 11 | AVNRT | N STUDY | 8 | 1.6 | 130 | 90 | 1 |
| KUMARI | 46 | F | 78 | 1 | 0 | 0 | 1 | 1 | 9 | AVNRT | N STUDY | 3.77 | 0.98 | 110 | 66 | 0 |
| LAKSHMI | 65 | F | 70 | 1 | 0 | 0 | 0 | 0 | 7.8 | AVNRT | N STUDY | 3.78 | 0.98 | 100 | 80 | 0 |
| MANGALAM | 54 | F | 70 | 2 | 0 | 0 | 1 | 1 | 8.2 | AVNRT | N STUDY | 3 | 0.46 | 180 | 90 | 1 |
| MANGAMMAL | 64 | F | 58 | 1 | 0 | 0 | 1 | 1 | 8.8 | AVNRT | N STUDY | 2.6 | 0.16 | 110 | 78 | 0 |
| MANI | 48 | M | 80 | 2 | 1 | 1 | 0 | 0 | 12 | AVNRT | N STUDY | 3.46 | 0.8 | 140 | 88 | 1 |
| MANOJ | 34 | M | 56 | 1 | 1 | 1 | 0 | 0 | 13.8 | AVNRT | N STUDY | 5.2 | 0.88 | 108 | 70 | 0 |
| MARIMUTHU | 70 | M | 65 | 1 | 0 | 0 | 0 | 1 | 11 | AVNRT | N STUDY | 5.68 | 1.08 | 142 | 88 | 1 |
| MARY | 64 | F | 68 | 1 | 0 | 0 | 1 | 0 | 9 | AVNRT | N STUDY | 6.8 | 0.36 | 138 | 90 | 1 |
| MUHAMED SIDIQ | 56 | M | 75 | 1 | 1 | 1 | 1 | 0 | 11.8 | AVNRT | N STUDY | 3.02 | 0.16 | 130 | 74 | 0 |
| MURUGADOSS | 78 | M | 68 | 1 | 1 | 1 | 1 | 1 | 7.8 | AVNRT | N STUDY | 5.8 | 0.26 | 170 | 90 | 1 |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| MURUGAMMAL | 90 | F | 54 | 1 | 0 | 0 | 1 | 1 | 7.3 | AVNRT | GRADE 1 DD | 10.08 | 0.8 | 80 | 60 | 0 |
| MURUGESAN | 65 | M | 68 | 1 | 1 | 1 | 1 | 1 | 5.6 | AVRT | WPW | 3.08 | 0.67 | 106 | 66 | 0 |
| NANDAKUMAR | 86 | M | 45 | 1 | 1 | 1 | 0 | 1 | 4.8 | AVRT | GRADE III DD | 2.06 | 0.08 | 108 | 70 | 0 |
| NARASIMHAN | 67 | M | 56 | 1 | 0 | 1 | 1 | 1 | 10.2 | AVNRT | GRADE I DD | 7.06 | 0.16 | 150 | 78 | 1 |
| NARMADA DEVI | 66 | M | 90 | 1 | 0 | 0 | 1 | 0 | 11-Jan | AVNRT | GRADEIV DD | 10.8 | 1.08 | 156 | 90 | 1 |
| PARVATHI | 54 | F | 86 | 1 | 0 | 0 | 1 | 1 | 9.8 | AVNRT | GRADE II DD 5.6 | 5.6 | 0.28 | 170 | 102 | 1 |
| POORNAM | 73 | F | 65 | 1 | 0 | 0 | 0 | 0 | 11 | AVNRT | N STUDY | 1.08 | 0.46 | 110 | 88 | 0 |
| PRABHA | 32 | F | 54 | 3 | 0 | 0 | 0 | 1 | 9.8 | AVNRT | N STUDY | 0.06 | 0.04 | 110 | 80 | 0 |
| RAJARATHINAM | 62 | M | 90 | 1 | 0 | 1 | 0 | 1 | 10.8 | AVNRT | GRADE I DD 2.8 | 2.8 | 1.06 | 180 | 90 | 1 |
| RAMANAIYA | 56 | M | 76 | 1 | 1 | 1 | 0 | 1 | 11 | AVNRT | GRADE II DD | 4.8 | 0.06 | 86 | 66 | 0 |
| RAMESH BABU | 49 | M | 68 | 1 | 1 | 1 | 0 | 1 | 13.2 | AVNRT | N STUDY | 2.96 | 0.24 | 140 | 90 | 1 |
| RATNAM | 46 | M | 46 | 1 | 1 | 0 | 1 | 0 | 8.8 | AVNRT | AS | 2.06 | 1.08 | 134 | 86 | 0 |
| RAVANAMMAL | 68 | F | 68 | 1 | 1 | 1 | 0 | 0 | 13.6 | AVNRT | GRADE I DD 4.800.36 | 4.8 | 0.34 | 130 | 90 | 1 |
| RAVINDRAN | 64 | M | 40 | 1 | 1 | 1 | 0 | 0 | 12.8 | AVNRT | N STUDY | 4.8 | 0.8 | 156 | 98 | 1 |
| SAMUVEL | 63 | M | 72 | 1 | 1 | 0 | 1 | 0 | 10.4 | AVNRT | N STUDY | 5.62 | 0.24 | 110 | 70 | 0 |
| SAROJA | 64 | F | 88 | 1 | 0 | 0 | 1 | 0 | 13.2 | AVNRT | GRADE 1 DD 4.90 | 4.9 | 2.8 | 180 | 90 | 1 |
| SELVARAJ | 63 | M | 66 | 1 | 0 | 0 | 0 | 0 | 11 | AVNRT | )E 1 DD 4.90 0.67 | 4.86 | 1.08 | 140 | 90 | 0 |
| SINGARI | 54 | F | 68 | 1 | 0 | 0 | 1 | 1 | 11 | AVNRT | N STUDY | 1.06 | 0.68 | 170 | 80 | 1 |
| SIVAGURU | 52 | M | 88 | 1 | 1 | 0 | 1 | 1 | 11.6 | AVNRT | N STUDY | 1.08 | 0.02 | 110 | 80 | 0 |
| SIVAN | 56 | M | 46 | 1 | 0 | 0 | 1 | 1 | 13 | AVNRT | N STUDY | 3.08 | 0.48 | 130 | 90 | 0 |
| SIVARAMAN | 46 | M | 68 | 1 | 0 | 1 | 0 | 0 | 11.6 | AVNRT | GRADE II DD | 5.6 | 0.06 | 66 | 40 | 0 |
| SRIDHARAN | 52 | M | 76 | 1 | 1 | 0 | 0 | 0 | 13 | AVNRT | E II DD 4.96 0.92 | 4.98 | 0.92 | 120 | 80 | 0 |
| STEPHEN | 48 | M | 78 | 1 | 1 | 1 | 0 | 1 | 11.2 | AVNRT | N STUDY | 4.26 | 0.04 | 136 | 88 | 0 |
| SUGUNA | 80 | F | 98 | 1 | 0 | 0 | 1 | 1 | 10.8 | AVNRT | AV SCLEROSIS 4.8 | 4.8 | 0.08 | 180 | 90 | 1 |
| SULTHAN | 66 | M | 78 | 1 | 1 | 0 | 1 | 1 | 11.8 | AVNRT | N STUDY | 9.36 | 0.82 | 130 | 80 | 0 |
| VINOTHINI | 78 | F | 68 | 2 | 0 | 0 | 1 | 1 | 6.8 | AVNRT | N STUDY | 0.46 | 0.08 | 110 | 80 | 0 |
| SURESH | 42 | M | 56 | 1 | 1 | 1 | 0 | 0 | 13.6 | AVNRT | N STUDY | 4.29 | 0.16 | 110 | 80 | 0 |
| THERESA | 70 | F | 56 | 1 | 0 | 0 | 0 | 1 | 11 | AVNRT | GRADE 1 DD 2.08 | 2.08 | 0.02 | 118 | 80 | 0 |
| VALLIAMMAL | 45 | F | 69 | 1 | 0 | 0 | 1 | 1 | 9.8 | AVNRT | N STUDY | 5.9 | 1.08 | 210 | 110 | 1 |
| VEENA | 14 | F | 45 | 1 | 0 | 0 | 0 | 0 | 11 | AVNRT | N STUDY | 3.06 | 0.42 | 100 | 70 | 0 |
| WILLIAMS | 82 | M | 60 | 1 | 0 | 0 | 0 | 0 | 8.8 | AVNRT | GRADE II DD 10.2 | 10.2 | 2.02 | 136 | 88 | 1 |

0 - Absence of Risk factor
1 - Persence of Risk factor

ETHICAL COMMITTEE
GOVT. KILPAUK MEDICAL COLLEGE, KILPAUK,
CHENNAI- 10.
Venue: PANAGAL HALL, KMC
Dt: 01.02.2011

CHAIRPERSON
Prof. Dr.V.KANAGASABAI, MD.,
Dean
Govt. Kilpauk Medical College, Chennai-10

Sub: Ethical Committee project work - approved – regarding.

Ref: Lr.No.3944/Audit/E1/09 Dt. 30.11.2010

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With above reference, the Institutional Ethical committee meeting for the following students was conducted at our Institution on 01.02.2011.

| S.NO. | Name | Topic |
|---|---|---|
| 1. | Dr.Navin Kumar, MS(Ortho), PG., Govt. Royapettah Hospital, Chennai. | 1.To Identify a Safe Zone to approach proximal Humerus<br>2.To study Anatomical relations of Axillary nerve, its course & its Variations |
| 2. | Dr.T.Satheesh Kumar, D.Ortho., PG., Govt. Royapettah Hospital, Chennai | Heriditary Multiple Exostosis |
| 3. | Dr.J. Jeya Shambavi, MD(Pathology), PG., Govt. Kilpauk Medical College, Chennai-10 | Clinicopathological Histomorphological and Immunohistochemical Study of Neuroendocrine Tumors of GIT |
| 4. | Dr.L. R. Saranya. MD., (Paed.)PG., Govt. Kilpauk Medical College, Chennai-10 | Cord Blood Zinc Level in Term-Small for Gestational Age Neonates |
| 5. | Dr.A.Satheesh Kumar, MS(ENT), PG., Kilpauk Medical College, Chennai | Study on Cases of Chronic Suppurative Otitis Media in Tubo Tympanic Type Due to Sinusitis as Focal Sepsis |
| 6. | R.Prathiban,(Msc.,Physiology), PG., Student, The TN. Dr.MGR Medical University,Chennai-32 | Prevalence of Cardiac Dysautonomia in Type I Diabetes mellitus |
| 7. | B. Manikandan, (Msc., Physiology), PG., Student, The TN Dr.M.G.R. Medical University, Chennai-32. | A Comparative Study of Left Ventricular Structure and Function in Obese and Non Obese Subjects |
| 8. | G. Selvakumar, (MSc., Physiology), PG., Student, The TN Dr.M.G.R. Medical University, Chennai-32. | A Study of the Intraocular Pressure In Patients with Diabetic Normotensive, Diabetic Hypertensive and Normal Subjects |

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|---|---|---|
| 9. | R. Ragulji, (Msc.,Physiology), PG., The TN Dr.MGR Medical University, Chennai-32. | A Study of Pulmonary function in insulin dependent diabetes mellitus |
| 10. | V.M. Jenila Veny,(MscPhysiology), PG. The TN Dr.MGR Medical University, Chennai-32 | Cardiovascular Autonomic Dysfunction in Chronic Kidney Disease |
| 11. | Dr.G. Lakshmi, MD(O&G), PG., Govt. Kilpauk Medical College,Chennai-10 | A Study of Association of Thyroid Disorders in Abnormal Uterine Bleeding |
| 12. | Dr.R. Harini, MD(O&G), PG., Kilpauk Medical College, Chennai | Single Dose Antibacterial treatment for Asymptomatic Bacteriuria in Pregnancy |
| 13. | Dr.E.Geetha, MD(O&G), PG., Govt. Kilpauk Medical College, Chennai-10 | A Study of the incidence course of Pregnancy and Pregnancy outcome in Obstetric Cholestasis and to evaluate the efficiency of UDCA in relieving the Symptoms and Improving the Perinatal outcome in these Patients |
| 14. | Dr.S. Nithya, MD(O&G), PG., Govt. Kilpauk Medical College, Chennai-10 | Prospective Study of Prevalence of diabetes Mellitus, Thyroid Dysfunction and Hyperprolactinemia in Recurrent Pregnancy loss |
| 15. | Dr.Mohideen Fathima, MD(O&G), PG., Govt. Kilpauk Medical College, Chennai-10 | A Study of evaluation of multi system changes in Gestational hypertension / severe pre-eclamptic/eclampasia patients |
| 16. | Dr.M.Padma Priya, MD(O&G), PG., Kilpauk Medical College, Chennai | Dyslipidemia as a Predictor of PIH |
| 17. | Mrs.G. Savitha, (Msc.,Medical Bio Chemistry), TN Dr.M.G.R.Medical University, Chennai-32. | Association of subclinical hypothyroidism in metabolic syndrome patients |
| 18. | Dr.K. Bharadhwaj, MD(G.M.), PG., Kilpauk Medical College, Ch-10 | A Study on Peripheral Vascular Disease in Type 2 Diabetes Mellitus |
| 19. | Dr.B.Priya, MD(G.M.), PG | Study of Serum Bilirubin Concentration in Established Coronary Artery Disease |
| 20. | Dr.R.Hema, MD(G.M.), PG., | Study of Troponin I level in Supraventricular Tachycardia in Non Cad Patients |
| 21. | Dr.P.Manoj Kumar, MD(G.M.), PG., Kilpauk Medical College, Ch-10 | A Study on Pulmonary Functions in Type 2 Diabetes Mellitus |
| 22. | Dr.M.Dhanasekar, MD(G.M.), PG., | Prognostic Risk Stratification of Acute Coronary Syndrome – Role of Highly Sencitive – Reactive Protien |
| 23. | Dr.N. Karthik, MD(G.M.), PG., Govt.Kilpauk Medical College, Chennai-10 | A Study of Comparison of QT Dispersion in Acute Myocardial Infraction Between Early Reperfusion and Late Reperfusion Therapy |

| | | |
|---|---|---|
| 24. | Dr.H. Anuradha, MD(G.M.), PG., Kilpauk Medical College, Ch-10 | A Study of Stress Hyperglycemia in Moderate Degree Burns |
| 25 | Dr. V. Nandakumar, MD(G.M.), PG., | A Prospective Study of Clinical Profile of Emphysematous Pyelonephritis in Type Two Diabetes Mellitus |
| 26. | Dr.S.Sasikumar, MS(G.S.), PG., Govt. Royapettah Hospital, Chennai | A Study of Unusual Presentations of Appendicitis. |
| 27. | Dr.S.R.Padmanabhan, MS(GS), PG., Govt. Royapettah Hospital, Chennai | A Comparative Study Between Autologous Platelet Rich Plasma and Saline Dressing for Diabetic Ulcer |
| 28. | Dr.C.Rose, Scientist-G and Head, Biotechnology, Central Leather Institute, Chennai. | Wound healing efficacy of the chitosan – containing collagenous biomaterial, on burn wound |
| 29. | E.K. Lavanya,B.Tech, Biotechnology, PG., Prathyusha Institute of Technology and Management, Tiruvallur. | Isolation and Characterization of Bacterial Pathogens from Eye Infection |

We are glad to inform you that at the Ethical Committee meeting, the document were discussed and the above short term projects are Ethically approved.

4/2/11

CHAIRPERSON

DEAN

Govt. Kilpauk Medical College,

Chennai-10.

To: The Individuals